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EDITORIAL

PROGNOSIS OF CORONARY THROMBOSIS

Coronary thrombosis looms large in the life of civilized man, be he physician or layman. It is emphatically a disease of civilization, striking the sedentary executive worker in the community, to whom stress, anxiety and obesity are occupational hazards. Upon what factors can a prognosis legitimately be based? It is now nearly 50 years ago that the first cases were described,[1] yet we still lack precise knowledge of the true factors that control the fate of the patient who has an attack of coronary thrombosis.

Recently Honey and Truelove[2] examined the records of the Radcliffe Infirmary, Oxford, and made a statistical analysis of the outcome of all cases of disease treated in the hospital between the years 1940 and 1954. There were 543 admissions which were beyond reasonable doubt (i.e. confirmed by electrocardiograph or post-mortem examination) of patients with acute attacks of coronary thrombosis. Progression was discussed in 3 phases, viz., first day, first 2 months (acute attack), and thereafter (long-term prognosis). Only one of the 543 cases was lost sight of. Honey and Truelove found that, of the 542, 62 (11%) had died within 24 hours of admission and 195 (35%) in the first 2 months. At least one more patient died within the succeeding 10 months, and yet another within the following year. Thereafter about 5% died each year. In this investigation Honey and Truelove examined the factors which have a bearing upon either the immediate or the long-term prognosis, as well as the influence of anticoagulant therapy upon prognosis.

In considering the deaths occurring in the acute attack, i.e. in the first 2 months, a number of factors were considered:

Age and sex: While the disease is twice as common in men as in women, the condition increases with advancing years and, since women appear to be affected at a later age than men, the fatality rate of the two groups was about the same. *Previous attack:* There was no appreciable difference in the mortality rate between the 88 cases with history of

VAN DIE REDAKSIE

PROGNOSE VAN KROONSLAGAARTROMBOSE

Kroonslagaartrombose neem 'n belangrike plek in die lewe van die beskaafde mensdom, of hy nou 'n dokter of 'n leek is. Dit is beslis 'n siekte van die beskawing, en tref die sittende uitvoerende werker in die gemeenskap vir wie spanning, angs en vetsug arbeidsrisiko's is. Op watter faktore kan 'n prognose met regverdigheid gebaseer word? Dit is nou amper 50 jaar gelede sedert die eerste gevalle beskryf is,[1] tog weet ons nog nie presies watter ware faktore die lot van die pasiënt, wat 'n aanval van kroonslagaartrombose het, beheer nie.

Onlangs het Honey en Truelove[2] die registers van die Radcliffe-Siekehuis, Oxford, nagegaan en 'n statistiese ontleding gemaak van die lot van alle gevalle van siekte wat tussen die jare 1940 en 1954 in die hospitaal behandel is. Daar was 543 toelatings wat sonder enige redelike twyfel (d.i. bevestig deur elektrokardiograaf en lykskouingsondersoek) van pasiënte met akute van kroonslagaartrombose was. Vooruitgang is onder 3 stadiums bespreek, nl. eerste dag, eerste 2 maande (akute aanval) en daarna (langtermyn prognose). Slegs een van die 543 gevalle is uit die oog verloor. Honey en Truelove het gevind dat van die 542, 62 (11%) binne 24 uur na toelating gesterf het en 195 (35%) binne die eerste 2 maande. Ten minste nog een pasiënt het binne die daaropvolgende 10 maande gesterf, en nog 'n ander een binne die volgende jaar. Daarna het ongeveer 5% elke jaar gesterf. By hierdie ondersoek het Honey en Truelove die faktore wat betrekking het op òf die onmiddellike òf die langtermyn prognose ondersoek, asook die invloed van stollingsverhinderende terapie op prognose.

By beskouing van die sterfgevalle wat gedurende die akute aanval voorgekom het, d.i. gedurende die eerste 2 maande, is 'n aantal faktore oorweeg:

Ouderdom en geslag: Terwyl die siekte tweemaal meer algemeen by mans as by vrouens is, neem die siekte met die ouderdom toe, en aangesien dit skyn of vrouens op 'n hoër ouderdom as mans aangetas word, was die noodlottigheidsyfer van die twee groepe ongeveer dieselfde. *Vorige aanval:* Daar was geen noemenswaardige verskil in die sterftesyfer tussen die 88 gevalle met 'n geskiedenis van

previous attacks and those admitted in their first episode. *Previous angina of effort:* Nearly 40% of the patients had suffered from angina, yet in neither sex was the fatality rate affected by it. *Previous hypertension* (i.e. pressures above 160 mm. Hg systolic or 100 diastolic): About one-third of the cases were known to have been hypertensive, yet this had no effect on the fatality rate. Nor did previous *dyspnoea on exertion* appear to be a factor, and it did not seem to matter whether the patient was at rest or exerting himself when the attack came on. If, however, the case had been admitted in a state of *clinical shock*, the prognosis was much worse, and if hypotension (i.e. diastolic pressure below 100 mm. Hg) was present as well, the prognosis was grave; 2 out of every 3 cases died within 48 hours. Other concomitant clinical features that appeared to have no influence upon the course of events in the acute phase were: pre-existing diabetes mellitus, cardiac asthma, heart failure, cardiac arrythmias, including extra-systoles, and cardiomegaly. To sum up, therefore, Honey and Truelove found that the most important factor determining whether or not a patient would survive the first 48 hours was the degree of circulatory shock present; other factors were unimportant.

But some of these factors do have a bearing on the long-term prognosis. If the patient survived the first 48 hours, an entirely different set of circumstances determined the risk of death. These were diabetes, cardiac asthma, heart failure, cardiac arrythmias, cardiomegaly, and—to a lesser extent—a history of a previous attack of myocardial infarction. Patients showing one or more of these features constituted a bad-risk group, and their chance of dying within the first year was 4 times that of the good-risk group (22·8% as compared with 5·6%). In general the good-risk group has twice as good a prospect of surviving the first year as the bad-risk cases (50·8% as compared with 27·8%).

The influence of anticoagulants upon the prognosis is slight, save only for an almost complete removal of the risk of pulmonary embolism. Since this complication accounts for about 6% of all deaths in patients surviving the first 48 hours, anticoagulants do exert some statistical benefit upon prognosis; when they were used the incidence of this complication was only 1%. Honey and Truelove estimated upon the basis of their investigations, that only 10 lives out of 263 could be regarded as saved by anticoagulant therapy. If this view is tenable, then Honey and Truelove's reassessment of the value of these preparations will constitute the most arresting feature of their report. If anticoagulants benefit only by preventing possible pulmonary embolism, clinicians will have to think again over the indications for their use. Other prognostic idols of ill omen that Honey and Truelove appear to demolish are a history of previous attack, angina of effort, dyspnoea on exertion, and hypertension—all of which, according to their statistical survey, have no apparent association with prognosis.

1. Obrastzow, W. P. and Straschesko, N. D. (1910): *Z. klin. Med.*, **71**, 116.
2. Honey, G. E. and Truelove, S. C. (1957): *Lancet*, **1**, 1161 and 1209.

vorige aanvalle en dié met 'n geskiedenis van 'n eerste aanval nie. *Vorige angina van inspanning:* Amper 40% van die pasiënte het aan angina gely, tog was die noodlottigheidsyfer by nie een van die twee geslagte beïnvloed nie. *Vorige drukverhoging* (d.i. drukkings bo 160 mm. Hg sistolies of 100 diastolies): Dit was bekend dat ongeveer een-derde van die gevalle aan drukverhoging gely het, en tog het dit geen uitwerking op die noodlottigheidsyfer gehad nie. Dit het ook geskyn of vorige *dispnee met inspanning* nie 'n faktor was nie, en dit het skynbaar nie saak gemaak of die pasiënt gerus het of homself ingespan het ten tye van die aanval nie. As die geval egter in 'n toestand van *kliniese skok* toegelaat is, was die prognose baie slegter, en as drukverlaging (d.i. diastoliese drukking onder 100 mm. Hg) ook aanwesig was, was die prognose ernstig; 2 uit elke 3 gevalle het binne 48 uur gesterf. Ander begeleidende kliniese kenmerke wat geblyk het geen invloed te hê op die gang van gebeurtenisse in die akute stadium nie, was: voorafbestaande diabetes mellitus, hartasma, hartondoeltreffendheid, hartaritmieë, insluitende buite-sistole, en kardiomegalie. Dus, om op te som, Honey en Truelove het gevind dat die belangrikste faktor wat bepaal of 'n pasiënt die eerste 48 uur sal oorleef of nie, die mate van sirkulerende skok aanwesig was; ander faktore was onbelangrik.

Maar sommige van hierdie faktore het wel betrekking op die langtermyn prognose. As die pasiënt die eerste 48 oorleef het, het 'n heeltemal verskillende reeks van omstandighede die risiko dat die pasiënt sal sterf, bepaal. Hulle was suikersiekte, hartasma, hartondoeltreffendheid, hartaritmieë, kardiomegalie en—tot 'n mindere mate—'n geskiedenis van 'n vorige aanval van miokardiese verstopping. Pasiënte wat een of meer van hierdie kenmerke getoon het, het die slegte risiko-groep gevorm, en hulle kanse om binne die eerste jaar te sterf, was 4-maal dié van die goeie risiko-groep (22·8% in vergelyking met 5·6%). Oor die algemeen was die vooruitsig om die eerste jaar te oorleef, 2-maal beter by die goeie risiko-groep as die slegte risiko-groep (50·8% in vergelyking met 27·8%).

Die invloed van teenstollingsmiddels op die prognose is gering, behalwe dat dit byna geheel en al die gevaar van longembolie uitskakel. Aangesien hierdie komplikasie verantwoordelik is vir ongeveer 6% van alle sterfgevalle by pasiënte wat die eerste 48 uur oorleef, oefen teenstollingsmiddels wel 'n sekere statistiese invloed op prognose uit; by gebruik daarvan was die voorkoms van hierdie komplikasie slegs 1%. Op die grondslag van hulle navorsings, het Honey en Truelove geskat dat dit beskou kan word dat slegs 10 lewens uit 263 deur stollingsverhinderende terapie gered is. As hierdie sienswyse geldig is, dan sal Honey en Truelove se herberaming van die nut van hierdie preparate die boeiendste kenmerk van hulle verslag vorm. Indien teenstollingsmiddels se enigste nut daarin lê om moontlike longembolie te verhoed, sal klinici op nuut aandag moet skenk aan die aanduidings vir hulle gebruik. Ander prognostiese wanbegrippe van rampspoed wat Honey en Truelove skynbaar omvergooi, is 'n geskiedenis van vorige aanval, angina van inspanning, dispnee met inspanning, en drukverhoging—almal waarvan, volgens hulle statistiese oorsig, geen skynbare verband met prognose het nie.

1. Obrastzow, W. P. en Straschesko, N. D. (1910): *Z. klin. Med.*, **71**, 116.
2. Honey, G. E. en Truelove, S. C. (1957): *Lancet*, **1**, 1161 en 1209.

MANAGEMENT OF ASTHMA AND HAY FEVER

Asthma is a common condition and probably commoner than is generally realized. It is said rarely to cause death, but probably more mortality is caused by asthma than death certificates reflect. The longer a patient has had the disease the more difficult does treatment become. Hence at the very first diagnosis investigation should be made to find a possible cause. Hospitals admit the worst cases and it is there that deaths more commonly occur. Complications from infection and deformities of the chest may be avoided if proper treatment is instituted early in life.

Among the 'types' of asthma are the allergic, infective, mixed allergic and infective, psychological, drug-sensitive, nasal (especially associated with polypi), and others (for example menstrual and exertional). Careful investigation may reveal the type. With early diagnosis, investigation and elimination of allergens (e.g. the in bedroom), infections and polypi, breathing exercises, and desensitization, good results may be obtained.

Patients with *status asthmaticus* should be admitted to hospital; the change in environment may in itself be helpful. If the older drugs (adrenaline, aminophylline) prove unsuccessful penicillin and the corticosteroid-type hormones are indicated. Cortisone has been administered orally; it must be given in big enough doses at 4-hourly intervals. The newer steroids are preferable, for example prednisone, which is less liable to disturb mineral metabolism—initially 50 mg., which is repeated, and then 10 mg. doses, at 4-hourly intervals. Corticotrophin, by injection, is preferred by some physicians. With these hormonal preparations care is required, withdrawal taking about 10 days. If maintenance doses of steroids need to be given, great care is required and, should infection occur, larger doses will be needed. Antibiotics such as penicillin, streptomycin, or tetracyclines may need to be used with the hormones given for the attack, in order to eliminate infection.

Hay fever is a condition that is often misdiagnosed, other illnesses such as coryza and vasomotor rhinorrhoea being mistaken for it; a number of antihypertensive drugs may produce nasal stuffiness or blocking of the nose, and other conditions must be considered. For the diagnosis of hay fever a careful history is required; the seasonal occurrence is an important feature.

About 10% of the population suffers from major allergic disorders and many more from minor ailments of allergic nature. The identification of allergens calls for a definite procedure. The history is important, and direct questioning is necessary (e.g. is the condition seasonal, or is it perennial?) In this way pollens or moulds may be established as allergens at particular seasons. Sneezing during spring cleaning, dusting, or contact with animals should be specifically inquired into. Skin testing is difficult to interpret and needs to be studied carefully by the expert; confirmation is obtained by the clinical course of the case. Good material is available from pollen and moulds for skin testing.

The vacuum cleaner is valuable in the home to control home dust and may reduce the frequency and severity of the symptoms. The housewife may wear a mask during house dusting. Pre-seasonal injection for desensitization may be performed, depending on the severity of the case. Treatment will need to be repeated year after year.

SOLITARY MASTOCYTOMA AND THE MASTOCYTOSES

A DISCUSSION OF THE MASTOCYTOSES AND A REPORT OF TWO CASES OF SOLITARY MASTOCYTOMA SHOWING AN UNUSUAL PHENOMENON OF GENERALIZED FLUSHING

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Tissue mast cells are connective-tissue cells of the histiocyte family whose cytoplasm contains large granules which stain metachromatically with such dyes as toluidine blue. They are found widely distributed through the connective tissue, especially in the walls of blood vessels and the immediately adjacent tissue. Normal human skin contains relatively few mast cells but their numbers are increased in a variety of pathological states such as itching dermatoses, lupus erythematosus, carcinoma, granulation tissue of healing wounds etc. Only in states of mastocytosis, of which urticaria pigmentosa is the classic example, are mast-cell infiltrates of diagnostic significance to be found.

All varieties of mastocytoses are rare and, until recently, were generally collected and described under the heading

of urticaria pigmentosa, the commonest clinical type and that first to be recognized. Urticaria pigmentosa, used in its broadest sense, describes certain related states of benign disseminate mastocytosis. On either side of this group are to be found the solitary (benign) mastocytomas and the malignant mastocytoses with *formes de passage* relating them to the central group.

Mastocytoma is a fairly common tumour of dogs, the solitary type being generally benign and multiple tumours malignant.[1] Solitary mastocytoma in man is rare, or perhaps rarely recognized. Chargin and Sachs,[2] in 1954 described 7 cases under the title 'Urticaria pigmentosa appearing as a solitary nodular lesion' and state that only 3 cases had previously been described in the (American) literature—those of Gross[3], Novy[4] and Scott and Lewis[5]. The literature before 1954 does, indeed, yield few references to the condition, but we can add the observations of Cuilleret,[6] of Costello,[7] of Bolgert and le Sourd[8] (who note that Civatte has seen some cases) and of Gaté, Terrier and Pruniéras.[9]

Since 1954 there have been further observations by Drennan and Beare,[10] de Graciansky *et al.*,[11] who refer to 7 previous cases in the French literature, Degos *et al.*[12] and Becker.[13]

The clinical appearances in solitary mastocytoma are remarkably constant. The lesion is present at birth or appears soon afterwards as a pink, red or yellow papule or small plaque on any part of the skin but with a certain predilection for the extremities, especially the wrist. For a few weeks or months the lesion may increase in size to reach its final state as a round or oval, well-circumscribed, reddish to light brown, slightly elevated plaque 1-5 cm. in diameter. The rounded surface may be smooth or have a *peau d'orange* appearance and a rubbery infiltration is palpable. At intervals of several weeks the tumour spontaneously, or after irritation, suddenly swells, becomes red and may develop superficial vesicles or bullae that rupture but soon heal. Rubbing of the quiescent lesion generally causes some turgescence which may be followed by vesiculation. Dermographism of the unaffected skin is demonstrable in some cases.

The sexes are equally affected and in no case so far described has any other family member been affected with mastocytosis. Multiplication of lesions does not seem to occur and, although time may bring a diminution in size, spontaneous cure has not been reported. In one case where the lesion was excised there was no recurrence in 17 years.[2, 3] No certain evidence of visceral mastocytosis has been found in those cases which have been fully investigated with this possibility in mind, but de Graciansky's case showed radiological signs of abnormalities in the lungs.[11] Diagnosis ultimately rests on the demonstration of a mast-cell infiltrate in the affected dermis. It is probable that many solitary mastocytomas are dismissed as moles or banal naevi—which, in their quiescent state, they closely resemble.

The two cases we have seen both presented a most unusual feature of generalized flushing accompanying spontaneous (but not induced) swelling of the mastocytoma. This flushing phenomenon has been noted only once before in a case of solitary mastocytoma by Degos,[12] and in de Graciansky's case[11] a generalized bullous eruption was seen on two occasions (separated by months) to accompany spontaneous swelling and bulla formation in the tumour. Similar episodes have very rarely been reported as occurring in cases of urticaria pigmentosa. Degos[14, 15] has seen it twice in cases of multinodular mastocytosis and refers to another instance reported by Morrow[16] in 1879. We are indebted to Professor Degos for this extract from Morrow's original description: 'An intense hyperaemia of the skin is occasioned by the least excitement. The child had no fear of me, yet when I proceeded to examine him the whole cutaneous surface became injected presenting the appearance of one suffering from a high fever.'

Wallace[17] has a patient with widespread urticaria pigmentosa who has flushing attacks and 'blackouts' every 2-3 months. Marten[18] describes a case of urticaria pigmentosa in a child in whom flushing attacks (lasting 25 minutes and occurring at long intervals) are accompanied by listlessness and rise in temperature. Bowers (in discussion of Marten's case) states that he has seen an infant with urticaria pigmentosa who flushes deeply and becomes drowsy when any lesion is rubbed. Dewar and Milne[19] report a case of bullous urticaria pigmentosa with repeated attacks of dyspnoea and cyanosis coinciding with fresh outcrops of bullae.

CASE REPORTS

Case 1

The patient was a healthy White girl aged 9 months with an oval (1×2 cm.), light-brown, slightly elevated plaque on the left lower abdomen. The overlying skin had a *peau d'orange* appearance and there was an elastic dermal infiltrate. This lesion was first noted as a pink papule when the child was 3 months old and was ascribed to irritation from adhesive plaster applied in the treatment of an umbilical hernia. Some increase in size and in depth of colour had been noted since its first appearance. At intervals of 5-6 weeks spontaneous swelling of the lesion occurred and it seemed as if the skin was on the point of blistering; but no vesicles ever formed and the swelling would subside in a day or two. The appearance and history suggested solitary mastocytoma but, on the first visit, friction did not elicit any swelling. Dermographism was not demonstrable on the normal skin.

Three weeks later the child was seen again about 12 hours after spontaneous swelling of the lesion had occurred. Swelling had increased its bulk so that it was elevated about 5 mm. over the surrounding skin, the surface was tense and whitish and the follicular orifices deeply indented. Onset of swelling had been accompanied by marked redness of the face (this redness stopped sharply at the neck and hair margin) and mottled erythema of shoulders and chest. No other symptoms were noted and skin colour had returned to normal within an hour.

A few days later, the swelling having subsided, a little local turgescence and erythema (but no generalized flushing) was produced by fairly vigorous pinching and rubbing, but much more trauma was required than is usually necessary to produce urtication of the lesions of urticaria pigmentosa in infants.

There was no enlargement of liver, spleen or lymph glands, and no abnormalities were discovered in the blood picture and bleeding time or by radiological examination of the skeleton. No other member of the family showed evidence of mastocytosis.

The tumour was excised in a quiescent state with a generous margin of normal skin and subcutaneous tissue. No flushing phenomena have been noted in the 6 months following operation; dermographism is not demonstrable.

Case 2

The patient was a healthy white boy aged 5 months with a roughly circular (1 cm. diameter), reddish-brown, elevated plaque on the inner aspect of the right arm near the elbow (Fig. 1). The lesion was present at birth and did not increase in size thereafter. Approximately every 3 weeks it became hard and swollen 'like a blister' and the child was subject to transient attacks, lasting 1-5 minutes, of flushing of the entire skin. The flush was bright red and only a few areas on the lower limbs were free; it lasted for about 5 minutes and then disappeared completely in one attack observed in hospital. No other symptoms were noted and the patient was apparently undisturbed. After 2-3 days the

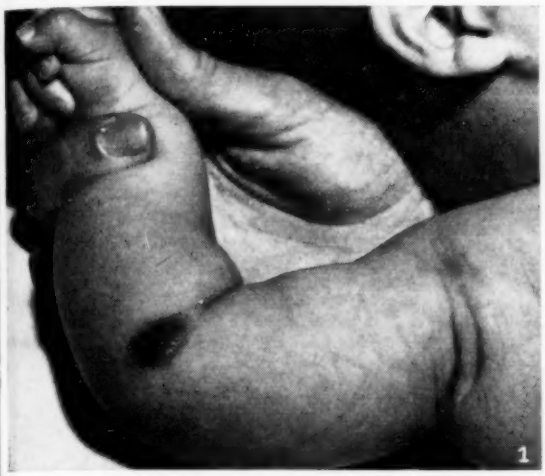

Fig. 1. Case 2. Solitary mastocytoma at right elbow.

blister burst and discharged a small amount of serous fluid; flushing attacks then ceased and did not recur until the lesion swelled again.

During quiescent periods the tumour could be made to swell by vigorous pinching and rubbing, and marked dermographism was at all times easily elicited on any part of the skin; the flush phenomenon only occurred spontaneously. No abnormalities were found in the blood picture, clotting time, prothrombin index, blood pressure or in the skeleton; liver and spleen were not enlarged. 5-hydroxy-indole acetic acid was not detected in the urine at a time when the lesion was quiescent. No other member of the family suffered from mastocytosis.

The tumor was excised and no flushing attacks occurred during the 2 months following operation: dermographism persisted.

HISTOPATHOLOGY

Case 1

The lesion measured 10 mm. in diameter and was raised about 1 mm. above the surrounding skin. The overlying epidermis showed no significant change in the stratum corneum, stratum granulosum or rete Malpighii. A few of the rete ridges were somewhat flattened. There was no evidence of vesiculation. Chargin and Sachs[2] found evidence of bulla formation in 7 out of 10 cases, in the majority of which it followed slight trauma or minor irritation. The basal cells contained more melanin pigment than those in the surrounding skin. No incontinence of pigment or phagocytosis by melanophores, as described by Degos,[20] was observed.

The central part of the tumour consisted of a mass of closely-packed mast cells traversed by a few thin strands of connective tissue. The mast cells extended up to the basal-cell layer (Fig. 2). Deeper down and at either side the cells were grouped in alveolar masses separated by bundles of collagen. At the periphery they

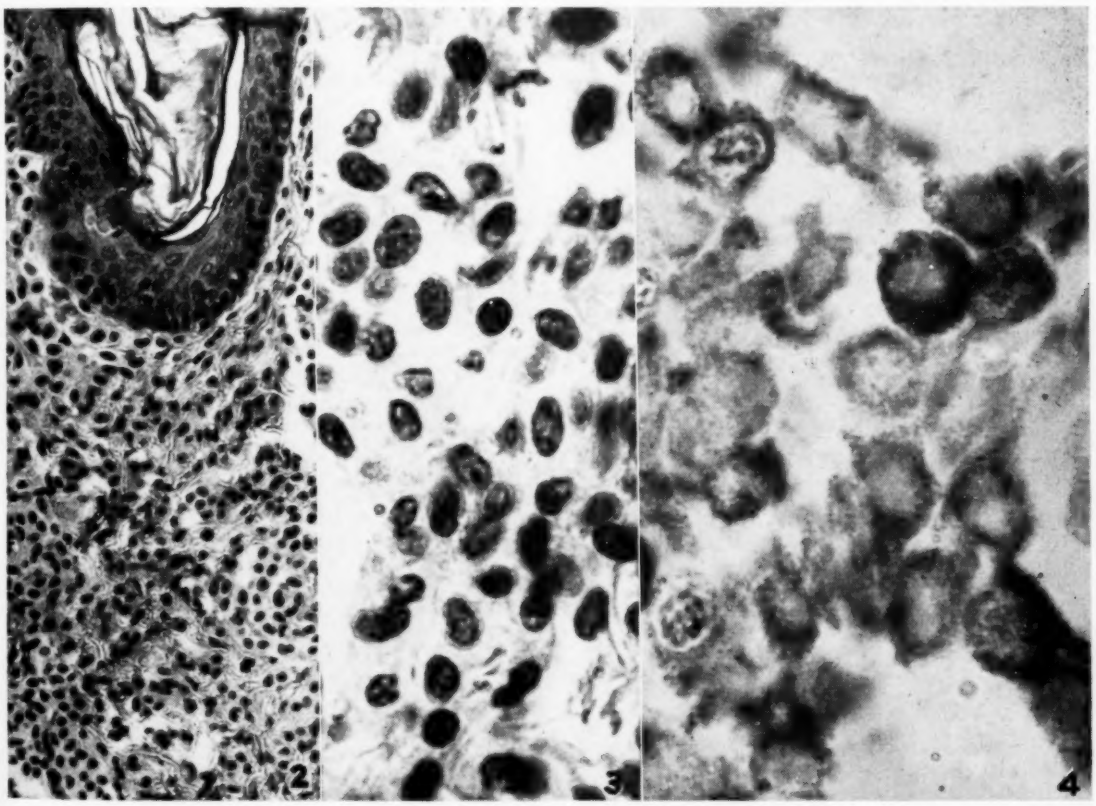

Fig. 2. Case 1. Massive cellular infiltrate extending up to the basal cells. H. & E. × 300. *Fig.* 3. Case 1. The infiltrate consists of a relatively uniform cell type with round or oval nuclei. H. & E. × 1,200. *Fig.* 4. Case 1. Metachromatic granules in the cytoplasm. Toluidine blue × 1,500.

were arranged around the blood vessels and hair follicles or in columns of single or double rows running between the collagen bundles. Surrounding the main lesion were scattered, isolated mast cells. In the central portion of the lesion the majority of the mast cells were round or oval (Figs. 3-4). A few were stellate and formed small syncytial masses. Among the isolated cells at the periphery a few were spindle-shaped. Chargin and Sachs[2] state that in the solitary nodular type of urticaria pigmentosa the infiltrate suggests that of a possible naevus or a lymphoblastoma, while in the macular type the cells resemble wandering connective-tissue cells, being more oval and smaller, and consisting chiefly of a nucleus.

No vacuolation of the cytoplasm was observed. Sagher[21] states that vacuoles appear after stimulation with histamine liberators.

An attempt was made to determine the age of the mast cells according to the description of Drennan and Beare.[10] The majority of them appeared to be mature, especially at the periphery of the lesion. In the upper part of the central tumour mass there were groups of maturing mast cells. These differed only in having a slightly larger, more vesicular nucleus and in the fact that the cystoplasmic granules were finer and stained less intensely with toluidine blue. In some cells the granules were orthochromatic. According to Sagher[21] the orthochromatic type is found mainly around blood vessels and is probably the earlier type of the fully developed metachromatic cell. Degos[22] maintains that Giemsa stain may show granules which are not demonstrable with toluidine blue. In our sections this phenomenon was well marked. Those cells regarded as maturing mast cells in toluidine-blue preparations stained as intensely with Giemsa as the mature cells. Neither mitotic nor amitotic division was seen.

Cottenot (cited by Degos[22] and Drennan[23]) both found that the granules of the mast cells in urticaria pigmentosa gave a positive reaction with P.A.S.* stain but the granules in normal tissue mast cells gave a negative reaction. In this case we found that the majority were P.A.S. positive.

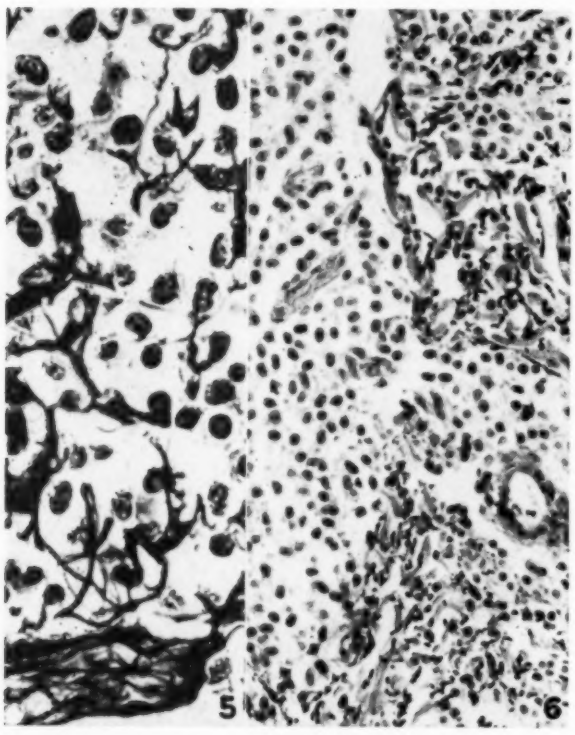

Fig. 5. Case 1. Fine reticulin network extending between the mast cells. Foote's stain. × 750.

Fig. 6. Case 1. Elastic fibres do not extend into the masses of mast cells. Verhoeff's stain. × 300.

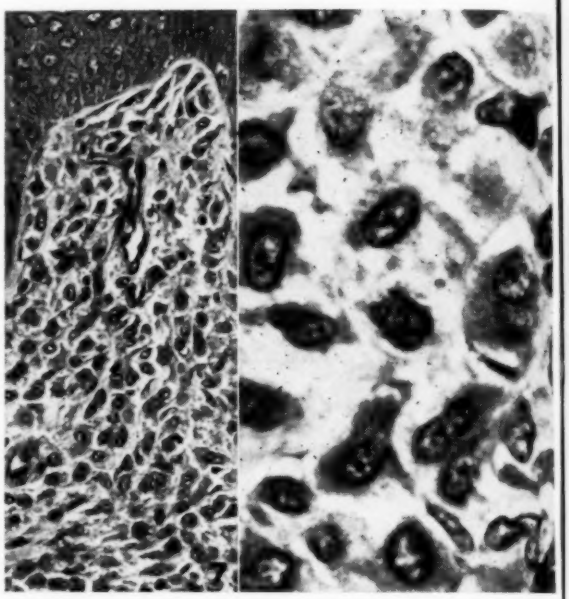

Fig. 7. Case 2. Massive cellular infiltrate extending up to the basal cells. H. & E. × 300.

Fig. 8. Case 2. The mast cells show marked variation in size and shape. Some of the nuclei are kidney-shaped. H. & E. × 1,200.

Drennan and Beare[10] found that some of the mast cells contained phagocytosed melanin pigment. In this case no pigment was visible in the haematoxylin-and-eosin sections, nor could any be demonstrated with Fontana's stain.

With regard to the stroma, Foote's silver stain showed that there was abundant reticulin between the mast cells. It surrounded individual cells or enclosed groups of 2-5 cells (Fig. 5).

Elastic tissue was found only accompanying the collagen bundles which separate the larger masses of mast cells (Fig. 6). A very occasional eosinophil leucocyte was present between the mast cells, but the eosinophil bodies described by Drennan and Beare[10] were not observed. Jadassohn (cited by Sagher[21]) maintained that eosinophils were seen in sections of urticaria pigmentosa skin lesions taken after irritation. Prakken and Woerdeman[24] described 2 cases of urticaria pigmentosa without eosinophils; neither of the lesions excised had been subjected to mechanical stimulation.

Case 2

The epidermis overlying the lesion showed no significant change apart from slight elongation and narrowing of the rete ridges. There was no vesiculation. There was a slight increase in melanin pigment in the basal cells. The papillae were somewhat enlarged.

In the dermis there was a massive infiltration by mast cells (Fig. 7). The distribution of these cells and the presence of reticulin and absence of elastic tissue between them was the same as in case 1.

No incontinence of melanin pigment was seen, chromatophores were absent, and no phagocytosis of melanin by mast cells could be demonstrated.

The mast cells were for the most part immature. They were stellate or spindle-shaped with an eosinophilic cytoplasm. Their nuclei were large and vesicular and many were bizarre in outline, taking the form of a kidney or a horse-shoe (Fig. 8). Several

* Periodic acid Schiff.

showed mitosis and many were undergoing amitotic division. Numerous small syncytial masses were present. With toluidine blue the majority showed only very fine orthochromatic granularity. Only at the periphery of the main mass, where the cells were lying separately, were there a few mature mast cells, round or oval in shape with smaller and darker nuclei and large metachromatic granules. As in case 1, many of the cells showing very fine orthochromatic granules with toluidine blue stained more intensely and showed larger granules with Giemsa stain. The majority of the granules were P.A.S. positive.

No vacuolation of the cytoplasm was seen. Neither eosinophil leucocytes nor eosinophil bodies could be found.

DISCUSSION

The structure and functions of the granules of tissue mast cells[25, 26] have been intensively studied in recent years but no clear picture of their role in normal and pathological states has yet emerged. It has been shown that mast cells contain heparin, histamine, 5-hydroxytryptamine and, probably, a precursor of hyaluronic acid (perhaps heparin), but it is still uncertain whether these substances are elaborated within the cells or simply stocked therein; and it is equally uncertain whether they exist separately or in combination. If biopsy is performed shortly after urtication has been induced in a lesion of urticaria pigmentosa, the number of granules in the mast cells is greatly reduced or they may even entirely disappear.[23, 27]

It is generally assumed that the *transitory* skin lesions of urticaria pigmentosa are due to the liberation of histamine or histamine-like substances from mast-cell granules. Urtication is inhibited by *high concentrations* of anthistamines. Waldenström *et al.*[28] investigated a case of urticaria pigmentosa and demonstrated histaminuria; a similar finding is noted by Hamrin,[29] who states that Pernow and Dunér have demonstrated this in 5 cases. Dewar and Milne[19] found the skin of an infant with bullous urticaria pigmentosa to contain almost 20 times the normal content of histamine, and suggest that toxic doses of antihistamines might be required to suppress urticating and bullous lesions. They could not reproduce bullous lesions by intradermal injection of histamine (or of heparin) in 2 (bullous) cases and doubt whether histamine release alone is the whole explanation of the skin manifestations in urticaria pigmentosa.

In our cases the generalized flushing may reasonably be assumed to be due to liberation in large quantity of the vaso-active substance which causes the local turgescence of the tumour. It is surprising that this flushing phenomenon should have occurred in 3 among the very small number of reported solitary mastocytomas while it has been described only very rarely in the voluminous literature (over 500 cases since 1869) on the diffuse mastocytoses, in which the total number of mast cells in the skin must be enormously greater. If generalized flushing is indeed largely bound to solitary mastocytoma it would place this condition a little apart from the other benign mastocytoses, which it otherwise greatly resembles.

In only one other condition—metastasizing argentaffinoma (carcinoid)—does generalized flushing comparable with that of mastocytoma occur. It is not suggested that this condition which, so far as we can discover, has never been described in infants, is likely to require consideration in differential diagnosis, but it is conceivable that the flush mechanism may be the same, or similar, in the two syndromes.

Carcinoid tumours[30, 31] arise from the argentaffin (Kultschitzky) cells of the gastro-intestinal tract; they generally occur at or after middle age (Becker's case[32] aged 15 is the youngest we have seen reported) and cause local symptoms such as abdominal pain, intestinal obstructions etc. as a result of their physical presence in the bowel. Carcinoid, if complicated by hepatic matastases, can cause general symptoms when a secretion of the argentaffin cells passes directly into the blood without being converted in the liver to an inactive metabolite.

The argentaffin cells are believed normally to constitute a diffuse endocrine gland and to manufacture 5-hydroxytryptamine (5HT, serotonin, enteramine) from tryptophan.[33] 5HT is a vaso-active substance and, like histamine, adrenaline and noradrenaline, appears to be absorbed, stored and released by the platelets; and as already noted, it seems to be a constituent of the granules of mast cells. The functions of 5HT in normal circumstances are not definitely established, but it has been suggested that it plays a part in haemostasis, regulation of vascular tone, control of peristalsis, maintenance of a normal central nervous system, and regulation of kidney function. 5HT is oxidized in the lungs to 5-hydroxy-indole-acetic acid (5HIAA), which is excreted in the urine. Confirmation of the diagnosis of metastasizing argentaffinoma may be obtained by estimation of the quantity of 5HIAA in the urine or of 5HT in serum or urine.

In argentaffinoma with hepatic metastases 5HT in excess passes into the general circulation and returns to the right atrium without being converted to inactive 5HIAA, and a peculiar syndrome results. The outstanding symptom is flushing of the face and upper part of the body; the flush may be a uniform erythema or a transient macular cyanosis, which may alternate with areas of erythema and pallor. Attacks of flushing may occur spontaneously or be induced by food, alcohol or pressure over the liver. Unlike those of mastocytoma, the flushes of carcinoid occur frequently and may even become constant and generalized, with the appearance of profuse, permanent telangiectases. Other symptoms are diarrhoea, tightness across the chest, and asthma. Changes eventually occur in the heart in those parts permanently bathed in an excess of 5HT, with the development of pulmonary or tricuspid stenosis (or both) and right heart failure. Pellagra-like symptoms have been noted and may be caused either by malnutrition induced by the diarrhoea or because 5HT production takes precedence over protein synthesis and nicotinic-acid formation.

We consider that the possibility that the vascular phenomena which occur in solitary mastocytoma and the mastocytoses generally, and in argentaffinoma, are due to the release of the same vaso-active substance, perhaps a histamine-5HT compound, deserves further investigation.

THE MASTOCYTOSES

Urticaria pigmentosa (Fig. 9) was described by Nettleship (1869) and named by Sangster (1878). Unna (1887) showed that mast cells predominated in the dermal infiltrate causing the lesions. Urticaria pigmentosa occurs classically in infants and children, lesions being present at birth or appearing soon afterwards. The course of the disease is marked by 3 phases. In the first (which may be omitted) there is a generalized eruption of pink or red urticarial maculopapules that increases by crops. After a few months the condition is stabilized in the second stage with yellowish-

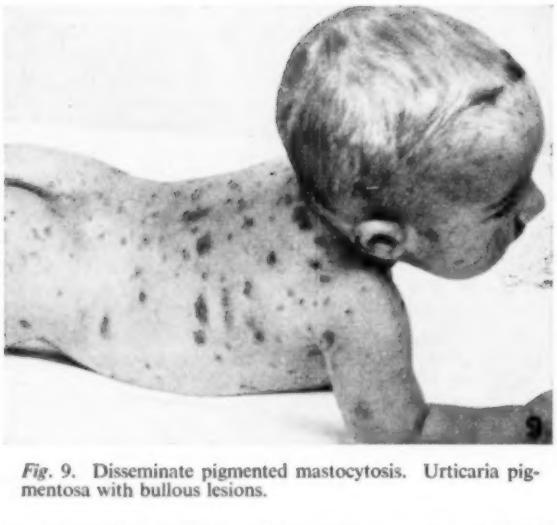

Fig. 9. Disseminate pigmented mastocytosis. Urticaria pigmentosa with bullous lesions.

brown maculo-papules or little round or oval, infiltrated plaques that urticate spontaneously or after friction. The phenomenon of induced urtication is commonly known as Darier's sign but was noted by the earliest observers. Dermographism is often demonstrable on normal skin between the lesions. The second stage lasts for some years and is followed by a final one in which urtication diminishes and the lesions flatten and finally disappear before the child reaches the age of puberty. Lesions are found mainly on the trunk and limbs, and the palms, soles, face, scalp and mucous membranes are rarely affected. Itch may occur sporadically but is seldom severe and the general health is unaffected.

This classical picture is seen often enough, but it is becoming increasingly clear that a great variety of clinical variants of mastocytosis may be encountered, that urticaria pigmentosa is only one of them, and that to continue to use the term urticaria pigmentosa in a broad sense is to employ a misnomer. The mastocytoses are unrelated to urticaria and their lesions do not always urticate and are not invariably pigmented. The concept of mastocytosis with urticaria pigmentosa as one manifestation is that of Sézary,[34] who expressed it in 1936, but long before this time there had been suggestions that urticaria pigmentosa was not a simple skin disorder but a disease, perhaps related to the leukaemias, in which visceral involvement such as lymphadenopathy, hepatosplenomegaly and discrete lymphoid or myeloid reactions in the blood might be encountered (Graham Little, and Jeanselme and Touraine, cited by de Graciansky and Paraf[35]).

There are many clinical varieties of mastocytosis apart from urticaria pigmentosa in childhood. A common one is that described by Tilbury Fox (1875) as xanthelasmoidea, in which the lesions are yellow papules, nodules or plaques that may suggest a diagnosis of xanthomatosis. This type is especially prone to undergo phases of spontaneous bulla formation, the bullae appearing on the existing lesions or on intervening normal skin; the tendency to bulla formation usually disappears after a year or two. In some otherwise typical cases of urticaria pigmentosa urtication does not occur; in others no mast cells can be found at biopsy, probably because the granules had been discharged just before the test was performed. The number of lesions in both the pigmented and xanthelasmoid types is very variable; sometimes they are few and discrete, sometimes closely aggregated, or even agglomerated into great plaques that spare little of the body surface. Occasionally the lesions are nodular or tumoral and very few in number, perhaps only two or three, and resembling the solitary mastocytomas. Very rarely a solitary, large, nodular lesion is associated with urticaria pigmentosa. Statements that urticaria pigmentosa of children generally disappears spontaneously were made, probably, on a basis of hope rather than observation, if we may judge from the many reported cases where the disease persisted into adult life.

Urticaria pigmentosa of adults (apart from that persisting from childhood) may begin at any age and remains indefinitely. Cases with a history of over 20 years are commonplace and some have had skin lesions for 50 years. Reputedly rare, personal observation and reference to the literature suggest that it is nearly as common as that of children. The lesions are usually little round or oval macules with no palpable infiltration, but papular forms also occur. Darier's sign may be elicited and spontaneous urtication may occur, but turgescence of the lesions is not so marked as in children. Dermographism may be demonstrable. Itch is unusual and bulla formation very rare. The general health is unaffected.

The condition described by Parkes Weber and Hellenschmied[36] as telangiectasia macularis eruptiva perstans, and characterized by multiple telangiectatic macules, is recognized as a relatively unpigmented variant of adult urticaria pigmentosa; *formes de passage* link the two. Vascular dilatation of more marked degree contributes to the type known as angiomatous urticaria pigmentosa. Marked telangiectasia of neck and cheeks is often a feature of urticaria pigmentosa in adults; this is another point in common with metastasizing carcinoid.

Mast-cell infiltrates in the dermis are characteristic of the histological picture of these mastocytoses. The infiltrate may be discrete and located particularly around the blood vessels (which may be dilated), the pilosebaceous follicles and the sweat glands or it may be dense and compact. Pigmentation is produced by increased melanin in the epidermis, which is otherwise unaltered. In specimens from lesions traumatized before excision there is oedema, an influx of eosinophils, and shrinking or even disappearance of mast cells and a great diminution in their granules. The cleavage line in bullous lesions is inconstant; in one case personally investigated (J.M.) the bulla was subepidermal and this was also the finding of Degos[14] and of Dewar and Milne,[19] but in other cases it was intra-epidermal or subcorneal.[2, 37]

Sézary *et al.*[38, 39] have shown that the phenomenon of dermographism is dependent on an increase in the number of dermal mast cells over the normal. Dermographism is a fairly common finding in the mastocytoses and indicates a more widespread involvement of the skin than might be suggested by the permanently visible lesions. Normal skin, according to Sézary, contains an average of 0·8 mast cells per field (6 objective, II ocular), and the threshold for the appearance of dermographism is 3·5 per field. There

were 45 mast cells per field in the abdominal skin of our case 2.

Within the last decade the concept of mastocytosis as a systematic disease has been consolidated by the accumulation of evidence that visceral involvement is not infrequent in chronic, benign cases and that malignant mastocytosis can occur.

Sagher *et al.*[40] in 1952 first drew attention to osseous lesions demonstrable radiologically in cases of urticaria pigmentosa. In 1956 Sagher[41] reports that 19 of 52 cases of urticaria pigmentosa showed radiological evidence of bone involvement. Lesions of two types may be found; (1) Generalized type with cystic osteoporosis of the ribs and thickening of osseous trabeculae, and osteosclerotic changes in the skull, pelvis and vertebrae. (2) Localized type with calcified deposits and decalcified areas in the humerus, radius, femur, skull and scapula. It has been shown at autopsy in one of Sagher's cases[41] that massive mast-cell infiltrates were present in the bones. Daly[42] also reports mast-cell granuloma at biopsy of bone in a case of urticaria pigmentosa with reticular osteoporosis in ribs and elsewhere.

De Graciansky *et al.*,[11] in their case of solitary mastocytoma with generalized bullous episodes, found on radiological examination of the thorax a micronodular infiltrate of the perihilar regions which might signify lung involvement by mast cells. Barker[43] reports on an adult with urticaria pigmentosa, bone lesions (decalcification), hepatosplenomegaly, lymphadenopathy, and a diffuse symmetrical reticulated pattern throughout both lungs.

Discrete lymphadenopathy and hepatosplenomegaly have for long been known sometimes to be associated with urticaria pigmentosa, and in one case of Jeanselme and Touraine (cited by de Graciansky and Paraf[35]) hepatosplenomegaly persisted over a period of 20 years. The presence of mast-cell infiltrates in the affected viscera has on several occasions been demonstrated by biopsy or puncture. In a few cases hepatic cirrhosis[44-46] has been found, alone or with mast-cell infiltrate, at autopsy or by puncture biopsy, and it has been suggested that heparin plays a role in the initiation of this process.

The blood and marrow pictures are almost always normal in the benign mastocytoses. We have mentioned the occasional observation of minor lymphoid or myeloid reactions in the blood, but tissue mast cells are not found in the circulation even in the rare cases when their presence has been demonstrated in the bone marrow. Basophil leucocytes of the myeloid series are unrelated to tissue mast cells and their numbers are unaffected in the mastocytoses. The association of mastocytosis with outspoken blood disorders has been noted on a few occasions, e.g. Sagher's case[41] of cutaneo-visceral mastocytosis, who died of monocytic leukaemia, ten Berg and Hermans' case[47] of haemorrhagic urticaria pigmentosa with paramyeloblast leukaemia, and Gatto's case (cited by de Graciansky and Paraf[35]) of urticaria pigmentosa with Cooley's erythroblastic anaemia. It is impossible to say whether the association of these disorders was significant or simply coincidental.

Although it might well be expected that the liberation of heparin from the mast-cell granules in states of mastocytosis would lead to some haemorrhagic tendency it is, in fact, rare for any abnormality in bleeding time, clotting time or prothrombin level to be discovered.[48, 49] Cases of mastocytosis with haemorrhagic lesions are extremely rare.[47, 50, 51]

Such visceral manifestations may be found with any of the cutaneous varieties of mastocytosis, with the possible exception of solitary mastocytoma, where positive proof of visceral lesions is still lacking. Their presence in no way alters the prognosis for the worse (ref. Jeanselme and Touraine's case with hepatosplenomegaly for 20 years[35]).

Malignant Mastocytosis

There have recently been described, however, a few cases of mastocytosis which differ greatly in clinical appearance and in outcome from the commoner, benign mastocytoses. The first fatal case of mastocytosis was that described in 1949 by Ellis[44] of a Negro child presenting greyish nodules in the skin at birth, who died of pulmonary oedema in a cachectic state at the age of 1 year. Portal cirrhosis was demonstrated at autopsy and mast cells were significantly increased in all tissues as compared with the normal figures.

In 1950 Hissard *et al.*[52] reported a case of cutaneo-visceral mastocytosis in an adult, in which the skin lesions (infiltration, nodules, ulceration) were like those specific infiltrates commonly found in the malignant reticuloses of all varieties. Mast cells were demonstrated in increased numbers in all tissues examined and were found in the circulating blood on many occasions, but in particularly high percentage after splenic puncture (47%) and after contraction of the spleen by adrenalin (15%). This case was further studied by Degos *et al.*[53] The patient eventually died. We have found only one other reference to a case in which mast cells were found in the blood, that of Efrati (cited by Sagher[21]).

Berlin,[54, 55] in 1955 described a third fatal case of cutaneo-visceral mastocytosis, in an old man. The skin lesions were suggestive of a reticulosis and mast cells were found in increased numbers in skin and viscera. This patient had suffered from attacks of diarrhoea for over 20 years, and we have found reports of 2 other cases[56, 57] with gastro-intestinal complaints and diarrhoea; the point is made to draw attention to yet another similarity between the symptoms of mastocytosis and those of carcinoid.

Degos and his colleagues[14, 22, 58-60] at the Hôpital Saint-Louis, Paris, have described other cases of cutaneous and cutaneo-visceral mastocytosis, differing materially from the benign forms, in which the clinical appearances are suggestive of malignant reticulosis. Béraud *et al.*[61] have reported on an infant with cutaneous mastocytosis in which the lesions (vesicles, crusted papules, purpura, pigmented poikiloderma, intertrigo, dermographism) suggested a diagnosis of Letterer-Siwe's disease. Hadida *et al.*[46] have seen an adult with a diffusely lichenified and pachydermatous skin and hepatomegaly; liver biopsy showed cirrhotic changes but no mast cells. Le Coulant and Texier[62] describe an adult with diffuse cutaneous infiltration, intense itch, lymphadenopathy and hepatosplenomegaly.

Course

Even in the malignant mastocytoses the course of the disease is often protracted and the suspicion of malignancy arises from the clinical appearance of the skin lesions and not from the microscopic picture, which offers no noteworthy differences from that of benign forms. Degos has seen more cases of malignant or potentially malignant mastocytoses (which he names *mastocytoses diffuses cutanées*) than any other observer and considers their symptomatology sufficiently characteristic to allow of their recognition even

in cases where no lesions of the urticaria pigmentosa type are present.[14, 22] The skin is ivory-coloured, thickened and infiltrated and may be smooth or sown with tiny granular papules; the natural furrows are exaggerated (Fig. 10). In minor forms thickening is minimal and colour change

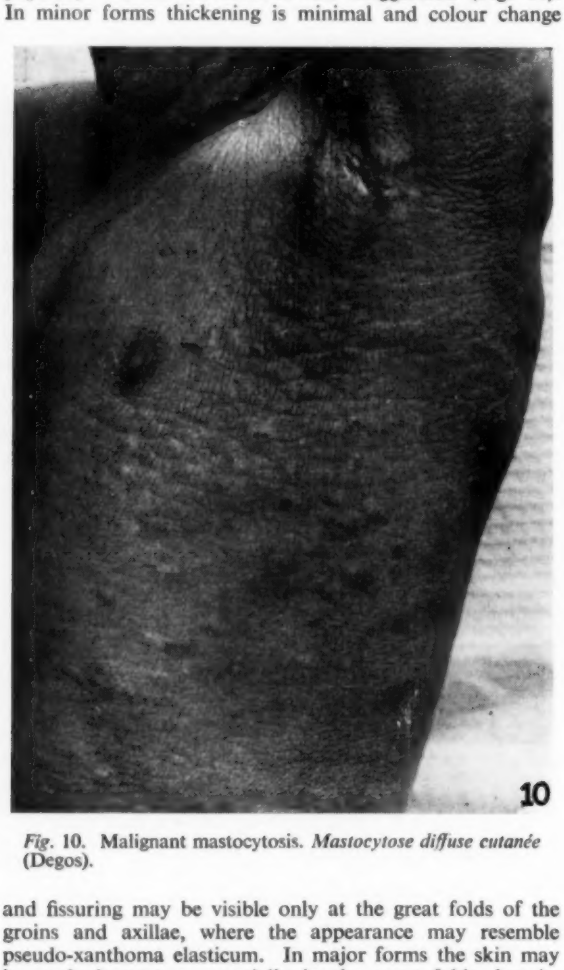

Fig. 10. Malignant mastocytosis. *Mastocytose diffuse cutanée* (Degos).

and fissuring may be visible only at the great folds of the groins and axillae, where the appearance may resemble pseudo-xanthoma elasticum. In major forms the skin may be pachydermatous, especially in the great folds, but its consistency is soft as opposed to the hardening felt in lichenification. Nodules, little tumours, bullae, excoriations and ulcerations may be seen. Large plaques of erythroderma, showing easily induced turgescence and associated with lesions of urticaria pigmentosa, occurred in 2 cases. Pruritus is intense and often accompanied by dermographism.

Clinical Classification

In Table I we propose a system of classification of the mastocytoses according to clinical appearances.

TABLE I. THE MASTOCYTOSES

I. *Benign Mastocytoses*

A. *Solitary Mastocytoma*

Variants: with or without dermographism.
with episodes of generalized flushing.
with episodes of generalized bulla formation.

B. *Disseminate Mastocytoses*

1. Apparently purely cutaneous mastocytoses

(*a*) In children

(i) Discrete nodular or tumoral mastocytoses (restricted number of large lesions).
Variants: with pigmented lesions.
with xanthelasmoid lesions.

(ii) Disseminate xanthelasmoid mastocytosis (=xanthelasmoidea).
Variants: with or without dermographism.
with episodes of general flushing.
with episodes of bulla formation, papular, nodular, confluent, etc.

(iii) Disseminate pigmented mastocytosis (= urticaria pigmentosa of children).
Variants: with or without dermographism.
with episodes of general flushing.
with episodes of bulla formation, showing no urtication, macular, papular, nodular, confluent, etc.

(*b*) In Adults

Disseminate pigmented mastocytosis (= urticaria pigmentosa of adults).
Variants: with or without dermographism.
with bullous episodes, macular, papular, angiomatous, haemorrhagic, telangiectatic etc.
telangiectasia macularis eruptiva perstans.

(*c*) Dermographism

Some cases of dermographism may represent occult mastocytosis.

2. Cutaneo-visceral mastocytoses

Any of above plus lesions of bones or lungs, lymphodenopathy, hepatosplenomegaly or mast cell infiltrates in marrow. Circulating tissue mast cells never demonstrated. Prolonged course with no clinical suggestion or malignancy.

II. *Transitional Mastocytoses*

Cases showing skin lesions like those of the malignant reticuloses generally (diffuse infiltration, erythroderma, nodules, tumours, ulcers) and, frequently, dermographism, urtication and bullous episodes but no visceral lesions. Prognosis guarded.

III. *Malignant Mastocytoses*

Cases with skin lesions of the transitional type but with visceral involvement and, perhaps, tissue mast cells in the circulating blood. Fatal termination.

Cause

The cause of the mastocytoses is unknown. Ellis's patient[41] was born to a mother who had gonorrhoeal cervicitis during pregnancy, and a few similar cases have since been discovered.[63] The concept of urticaria pigmentosa as a naevoid disorder was suggested by the numerous cases in which the lesions are present at birth or appear shortly afterwards and by its occasional occurrence in uniovular twins or in families.[64-66] Now that it has been proved beyond doubt that the mastocytoses are systemic diseases it is clear that the earlier suggestion of a relationship to the leukaemias was, indeed, not unreasonable. Mast cells are, however, of the reticular series and their discovery in the circulation in states of mastocytosis is exceptional, so that the mastocytoses must be classified with the reticuloses and not with the leukoses. For Sézary,[67] the mastocytoses fit into his group of orthoplastic reticuloses which are characterized by proliferation of cells of types like those in simple inflammatory reactions, e.g. histiocytes, monocytes, lymphocytes, epithelioid cells, giant cells, normal plasmocytes,

mastocytes and fibroblasts. The group further includes sarcoidosis, Kaposi's acrosarcomatosis, and plasmocyte and eosinophil reticuloses. Orthoplastic reticuloses are generally benign, insensitive to radiotherapy and of slow evolution.

Treatment

A great variety of remedies has been tested against the mastocytoses (e.g. bismuth, sedatives, glandular extracts, heparin antagonists, infiltrations with hyaluronidase, steroid hormones, radiotherapy), all without lasting effect. The antihistamines are reported sometimes to give symptomatic relief but have proved useless in cases we have treated.

CONCLUSIONS

It seems reasonable to accept Sézary's classification[67] of the mastocytoses within the group of reticuloses. The frequency with which visceral lesions are demonstrated makes us wonder whether the mastocytoses are ever purely cutaneous diseases and we have reflected this doubt in the headings of our scheme of clinical classification. It seems probable that cases of mastocytosis showing visceral lesions, but negligible skin lesions or none at all, will be discovered, and we suggest that some cases of dermographism may deserve fuller investigation than they generally receive. Waldenström *et al.*[28] recently reported the case of an elderly woman who presented signs reminiscent of visceral mastocytosis and suffered repeated attacks of flushing like those seen in argentaffinoma. In this case there had for years been radiological evidence of infiltrates in lungs and bones. Histamine and 5HT were detectable in abnormal quantities in the blood and urine and 5HIAA excretion was markedly increased. Biopsy of bone showed an infiltrate of unidentified cells; no argentaffin cells were found by special staining techniques and the histamine content of the tissue was not high. No skin lesions suggestive of mastocytosis were present. The case is presented in the article as one of metastatic carcinoma but the primary tumour was undiscovered. Urticaria pigmentosa was considered in differential diagnosis but was dismissed. It is nevertheless tempting to think that this might possibly be a case of purely visceral mastocytosis.

The transitory cutaneous phenomena that characterize the mastocytoses (urtication, local erythema, bulla formation, dermographism) must be ascribed to the release, spontaneous or induced, of vaso-active substances from mast-cell granules, but it has not been proved that histamine alone is involved in the production of all or any of them. There is a striking similarity between the attacks of general flushing occasionally seen in mastocytosis and the flushing in cases of metastasizing carcinoid; the common factor may be 5HT, which occurs in both mast cells and argentaffin cells. It may be purely coincidental that permanent telangiectasia and diarrhoea, common symptoms of metastasizing carcinoid, are found sometimes with mastocytosis.

Another unsolved problem is the relationship of the fairly common benign mastocytoses to the malignant or potentially malignant mastocytoses. The latter show skin changes quite distinct from the former but seem to be accompanied at times by the same clinical manifestations of visceral involvement. A reliable means of differentiating the one from the other is required as a guide to prognosis; it may come from histological and biochemical studies.

SUMMARY

Two cases of solitary mastocytoma presenting episodes of generalized flushing are described. The nature of the flushing phenomenon is discussed and compared with that observed in metastasizing argentaffinoma. The question of mastocytosis in general is reviewed and a system of clinical classification and nomenclature is proposed.

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DIAGNOSIS OF FREE FLUID WITHIN THE PARANASAL SINUSES

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It is customary to diagnose 'free' fluid radiologically within the paranasal sinuses according to the rule that all fluid levels are horizontal and remain so whichever way the head is tilted in the erect position.[1] This generalization of fluid-level behaviour, its diagnostic dependability, and the customary tilt-testing technique for verification leave much to be desired.

THE CONCEPT OF 'INVARIANT HORIZONTALITY': IS IT TRUE?

A patient presented himself complaining of 'a cold that refused to get better'. Anteriorly the nose was clear, but naso-pharyngoscopically a stream of pus could be seen issuing from each middle meatus. He was referred for X-ray examination of the sinuses. In the erect occipito-mental view an opaque shadow, with unequivocally oblique upper limit, could be seen occupying the lower half of the left antral field (Fig. 1). The radiologist, an experienced observer, did not consider the left antral appearances presumptive evidence of free fluid. Tilt tests were nevertheless carried out. The erect tilt-to-the-right produced an undulant horizontal level (Fig. 2), that to the left a flat horizontal level (Fig. 3). The right and left recumbent occipito-mental positions both resulted in flat horizontal levels (Figs. 4 and 5).

A case with bilateral antral infection was filled by displacement in Parkinson's lateral low-head position with a penicillin-hyaluronidase aqueous opaque solution. X-ray in the right recumbent occipito-mental position revealed two separate horizontal fluid levels on different planes within each maxillary field (non-communicating, bilateral antral duplication) and a horizontal level within the right frontal sinus (Fig. 6). In the submento-vertical view the right antral field presented a frankly horizontal level, the left an unequivocally oblique or non-horizontal level (Fig. 7).

During an investigation of lead EDTA for contrast radiography within the paranasal sinus field,[2] the healthy sinuses of a technical assistant were filled by displacement with a 10% solution of lead EDTA in normal saline. Both antra and the left frontal sinus showed horizontal levels, and the right frontal sinus an oblique level, in the erect occipito-mental view (Fig. 8). In the right recumbent occipito-mental position X-ray revealed horizontal fluid levels in both antra, the right frontal sinus, and the main portion of the left frontal sinus cavity. The small loculus adjacent to the main portion of the left frontal sinus cavity showed an unequivocally oblique fluid level (Fig. 9).

A 35-year-old business man presented himself complaining of nasal obstruction, nasal discharge, and left supraorbital headaches. Examination revealed signs of bilateral antral and left frontal sinus suppuration. He was referred for X-ray examination. The erect occipito-mental view showed a clouded right antrum, a left antrum with concave horizontal fluid level and thickened mucosa, and a left frontal sinus containing an undulant horizontal fluid level (Fig. 10). Both fluid levels remain horizontal in the right recumbent occipito-mental position Fig. (11), but not in the left recumbent occipito-mental position. In the latter position both levels are now unequivocally oblique or non-horizontal in character (Fig. 12).

The occurrence of non-horizontal pathological fluid levels in different erect positions in the same sinus, together with the radiopaque reproduction and verification of horizontal and oblique (non-horizontal) fluid levels in one and the same sinus, and in companion sinuses, at one and the same time (i.e., in the same radiographic position) is

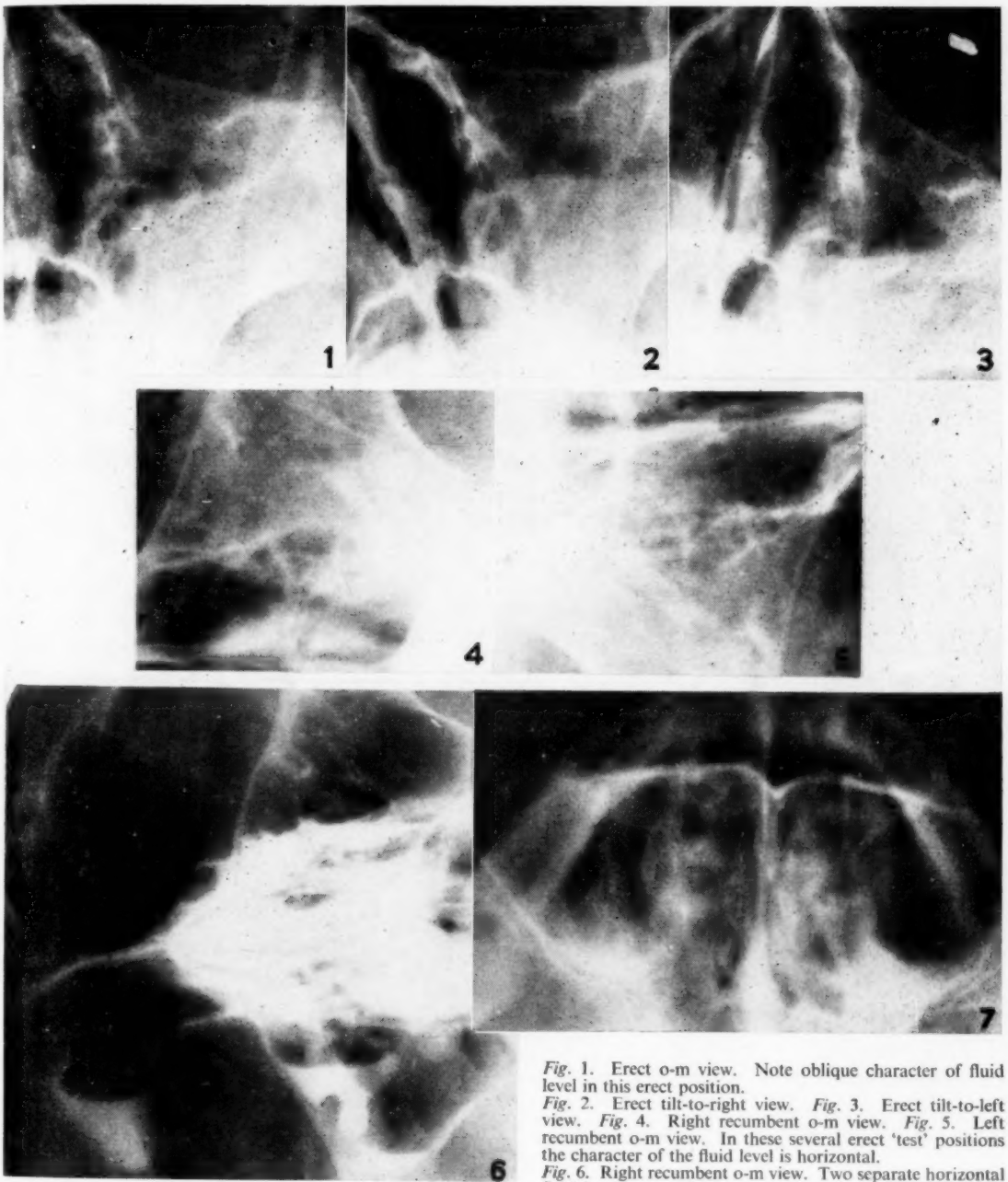
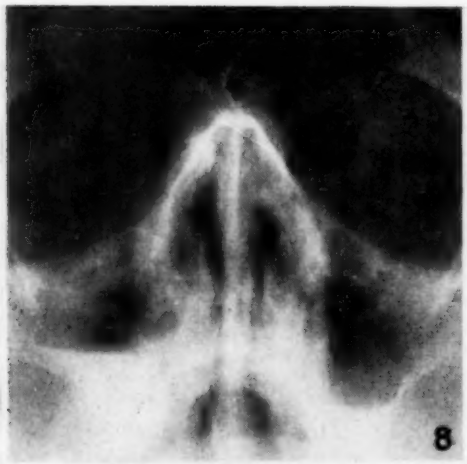
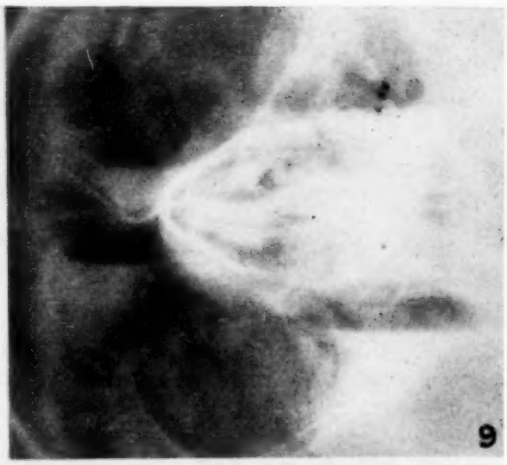
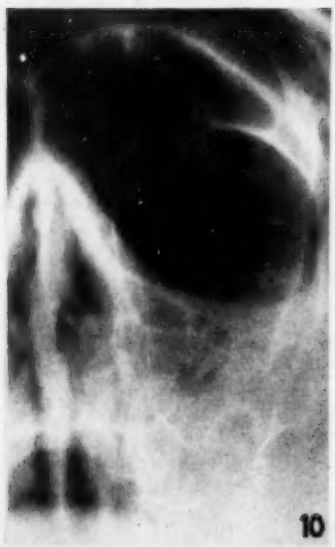
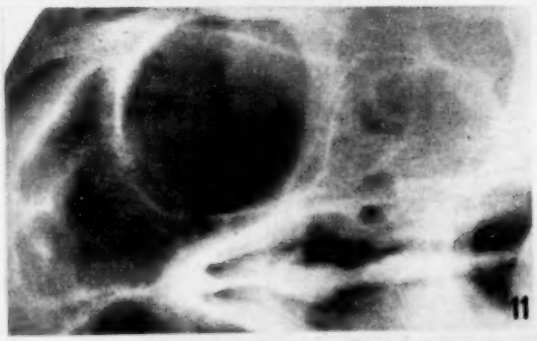


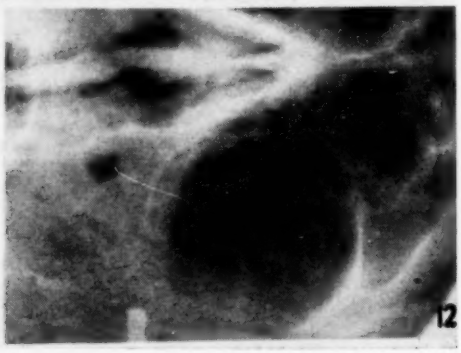
Fig. 1. Erect o-m view. Note oblique character of fluid level in this erect position.
Fig. 2. Erect tilt-to-right view. *Fig.* 3. Erect tilt-to-left view. *Fig.* 4. Right recumbent o-m view. *Fig.* 5. Left recumbent o-m view. In these several erect 'test' positions the character of the fluid level is horizontal.
Fig. 6. Right recumbent o-m view. Two separate horizontal fluid levels, on different planes, can be seen within each maxillary field (non-communicating, bilateral antral duplication). The fluid level in the right frontal sinus is also horizontal.
Fig. 7. Submento-vertical view. In this erect position only the fluid level in the right antral field shows the characteristic of 'horizontality', and the level in the left antrum is unequivocally 'oblique' or non-horizontal in character.

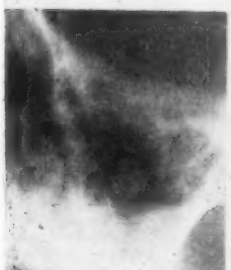












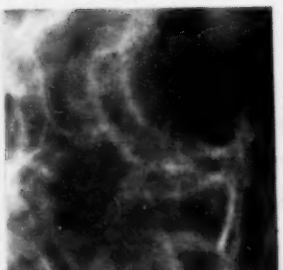


Fig. 8. Erect o-m view. Fluid levels in both antra and left frontal sinus are horizontal, that in right frontal sinus oblique.

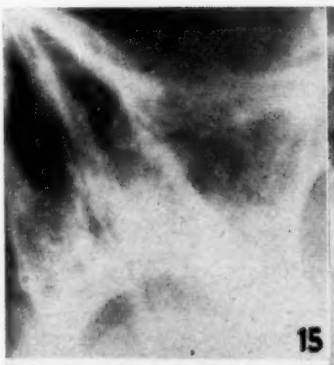
Fig. 9. Right recumbent o-m view. Levels in both antra still horizontal. The fluid in right frontal sinus now presents a horizontal level too. Left frontal sinus presents a fluid pool with horizontal level in main portion of sinus cavity, and one with non-horizontal or oblique level in adjacent small loculus.

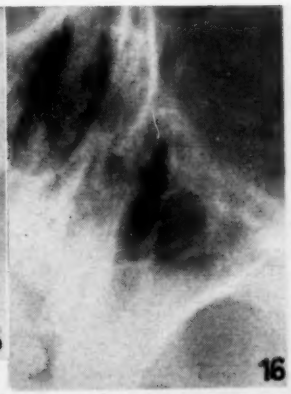
Fig. 10. Erect o-m view. Fluid levels in left antrum and left frontal sinus both horizontal in character in this erect position.

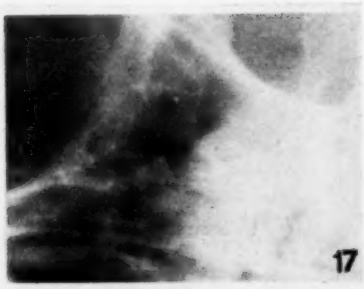
Fig. 11. Right recumbent o-m view. The characteristic of 'horizontality' is reproduced in fresh erect 'test' position.

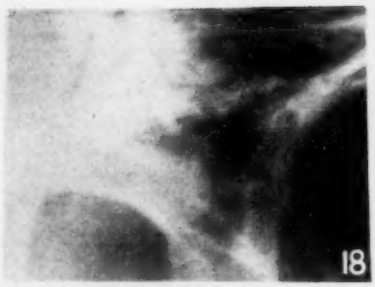
Fig. 12. Left recumbent o-m view. Characteristic of 'horizontality' not present in new erect 'test' position. Both fluid levels unequivocally oblique or non-horizontal in character.

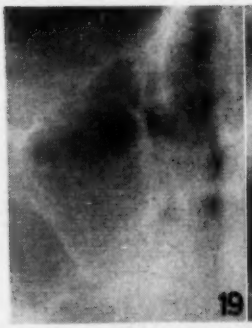
Fig. 13. Erect o-m view. *Fig.* 14. Erect lateral view. Opacity of lower half of left antrum with horizontal upper limit (level). 'Presumptive' evidence of pooling and collection of 'free' fluid.

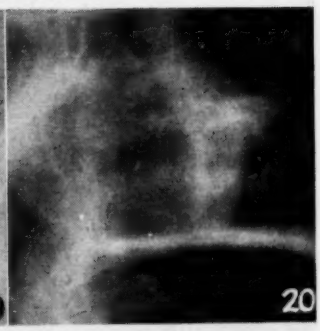


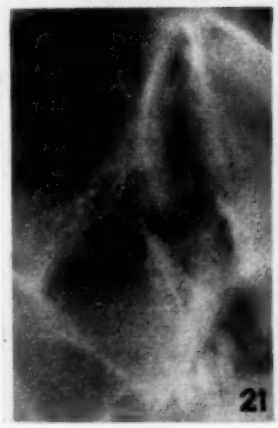












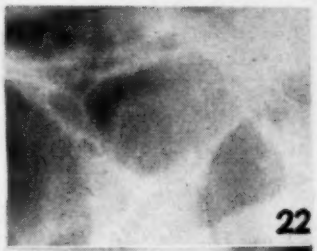


Fig. 15. Erect tilt-to-right view. *Fig.* 16. Erect tilt-to-left view. Reproduction of 'horizontality' by the customary partial-tilt test, currently accepted as diagnostic of 'free' fluid.

Fig. 17. Right recumbent o-m view. *Fig.* 18. Left recumbent o-m view. Complete right-angle tilt-testing positions. A more exacting test of 'free-ness' and the 'physical composition of shadow'. Note the finger-shaped shadow with blunt rounded end, as it lies first along the medial and then along the lateral antral wall in these respective positions. The opacity falls well short of the antral roof, is 'anchored' and not 'free', and presents anything but fluid level outlines.

Fig. 19. Erect o-m view. *Fig.* 20. Erect lateral view. Opacity of lower part of right antrum with horizontal upper limit (level). 'Presumptive' evidence of pooling and collection of 'free' fluid.

Fig. 21. Erect tilt-to-right view. Note reproduction of characteristic of 'horizontality' by customary partial-tilt test, generally accepted as diagnostic of 'free' fluid.

Fig. 22. Right recumbent o-m view. *Fig.* 23. Left recumbent o-m view. Complete right-angle tilt-testing positions. A more exacting test of 'free-ness' and of the 'physical composition of shadow'. Note the opacity with oblique upper border and blunt rounded end, well short of the antral roof, lying along the lateral and medial antral walls in these respective erect positions. The opacity is 'anchored' to the antral floor, i.e. not 'free', and does not present fluid level outlines.

Fig. 24. Submento-vertical view. Dependent portion of right antral field is unoccupied and radiolucent, thus substantiating absence of 'free' fluid.

Note: o-m is abbreviation for occipito-mental.

clear proof that sinus fluid levels are not invariably horizontal in all erect positions of the skull.

Since the character of a fluid level does not depend upon the radiologist's expectations (preconceived ideas) but upon the conditions at the level of pooling and collection, to claim that a fluid level 'will remain horizontal' is to defy nature and cast the radiologist in a Canutian role. The latter is as unenviable as the former is unscientific.

It is impossible to overlook the fact that paranasal sinuses are morphologically unpredictable and pathologically variable. Sinus space in consequence is not a fixed, uniform, standardized, and immutable entity. In such circumstances it is not possible to predetermine fluid-level behaviour.

And so the concept of 'invariant horizontality' is at variance with anatomy, pathology, and physical laws. As a description and prediction of fluid-level behaviour within the paranasal sinus field it fails to withstand both the test of direct observation and that of experimental verification. The conclusion is therefore inescapable that from the standpoint of practical diagnosis such a generalization is inadequate and should be discarded.

THE CUSTOMARY TILT-TEST FOR 'PROVING' FREE FLUID: IS IT DEPENDABLE?

A 45-year-old business man presented himself complaining of a recurring, burning pain in the left supraorbital region, which extended as far back as the ipsilateral parieto-occipital area. The pain was present on waking, disappeared soon after rising and had no accompaniments, and there was no local tenderness. Examination revealed a slightly tender left submaxillary gland, and a thick, 'catarrhal' stream seen coming from a pale, but otherwise normal-looking, left middle meatus on nasopharyngoscopic examination. Sinus X-ray study revealed in the erect occipito-mental, and left lateral views (Figs. 13 and 14) an opacity of the lower half of the left antral field with horizontal upper limit (presumptive evidence of 'free' fluid). Erect tilt-to-the-right and tilt-to-the-left views (Figs. 15 and 16) reproduced the characteristic of horizontality, thus apparently 'proving' the presence of 'free' fluid within the left antrum. As the result of experience gained with aqueous opaque media, and inflammatory sinus collections, during a clinico-diagnostic study of antral duplication,[3] the author has come to place much greater reliance on the recumbent occipito-mental position (view) for the verification of 'free' fluid, and the analysis of the 'physical composition of the shadow', within the sinus field. Right and left 'recumbent' views were accordingly done (Figs. 17 and 18). What had seemed to be 'free' fluid was now shown to be a non-tense, non-secreting cyst of the left antrum. No 'free' fluid whatsoever was present in this cell.

An adolescent Coloured girl presented herself complaining of unilateral nasal obstruction, and bilateral mucopurulent nasal discharge. X-ray of the sinuses revealed among other things an opacity of the lower half of the right antrum, with concave horizontal upper limit, in the erect occipito-mental view (Fig. 19). The erect lateral view (Fig. 20) showed a flat horizontal level extending from the anterior to the posterior wall of the right antrum (presumptive evidence of 'free' fluid). An erect tilt-to-the-right reproduced the characteristic of horizontality, the analytical transformation currently considered diagnostic of 'free' fluid within a sinus (Fig. 21). Following the author's standard practice, right and left recumbent occipito-mental views were also done (Figs. 22 and 23). What could have passed for 'free' fluid could now be clearly seen to be a non-tense, non-secreting cyst of the right antrum. No 'free' fluid whatsoever was present in the right antrum. This was substantiated by the submento-vertical view which showed a right antral field devoid of any circumscribed opacity in its dependent part (Fig. 24).

As non-tense sinus cysts may present a horizontal upper limit, which the customary partial tilt can reproduce, it is obvious that partial tilts may lead to misleading conclusions The complete right-angle tilt is a far more exacting test of 'free-ness', and of the 'physical composition of the shadow' in general within the sinus field. For example, shadows may consist of 'free' fluid plus submerged, radiologically indistinguishable, 'fixed' elements (mucosal swelling, cyst, polyp, neoplasm) and 'mobile' elements (mucino-cellular clump, nucleoprotein mass, blood clot, protein gel). It would be logical, therefore, to employ such tilts, rather than partial ones, in all cases presenting 'presumptive evidence' of 'free' fluid in erect views of the paranasal sinuses.

It is universally agreed that correct therapy depends upon accurate diagnosis. As reality is elusive, and only knowledge of nature can give the doctor mastery over it, the conclusion is inescapable that more realistic ideas are required regarding fluid-level behaviour, and free-fluid diagnosis within the paranasal sinuses.

SUMMARY

1. Attention is drawn to the fact that the concept of 'invariant horizontality' fails to describe and predict fluid-level behaviour within the paranasal sinuses, that it therefore represents an imperfect generalization of fluid-level behaviour within this field, and is thus diagnostically inadequate.

2. It is noted that fluid-level behaviour cannot be predetermined within the morphologically unpredictable and pathologically variable paranasal sinuses. Sinus space is not a fixed, uniform, standardized, immutable entity.

3. Attention is drawn to the fact that the customary partial tilt for the verification of free fluid in sinuses can be misleading. The suggestion is made that the complete right-angle tilt should be employed as a routine as a more exacting test of the characteristic of 'free-ness' and the 'physical composition of shadow.'

4. Illustrative cases are shown.

The writer wishes to thank Drs. A. A. Meyer, E. van der Burgh, S. J. Sarif, and K. V. O. Gunn for their radiological cooperation and assistance.

Special thanks are due to Dr. E. van der Burgh, and Prof. J. N. Jacobson of the Department of Radiology, Groote Schuur, Hospital, Cape Town, for their personal interest and criticism.

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STEP-FRACTURE OF THE DISTAL END OF THE RADIUS

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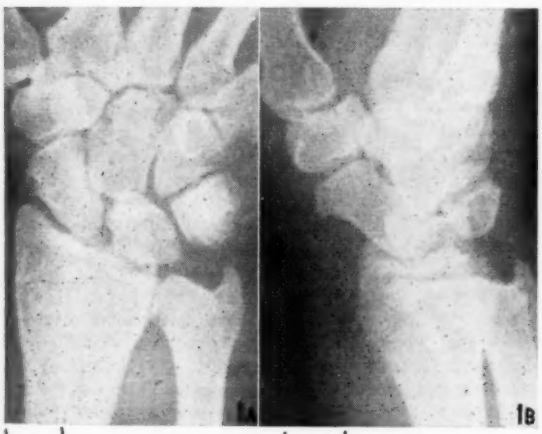
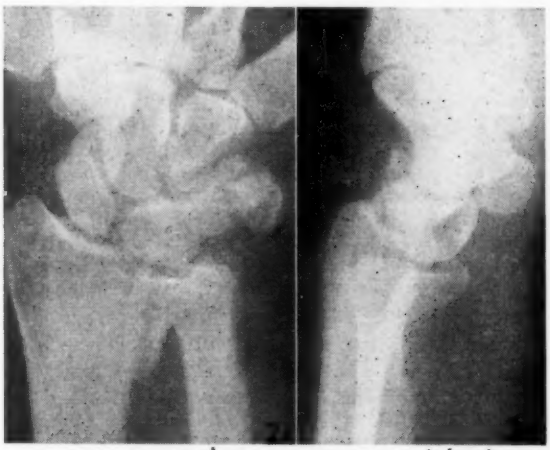
Fractures of the distal end of the radius occur commonly. In children pure epiphyseal displacement or epiphyseal displacement plus metaphyseal fragment are encountered in addition to greenstick fractures of the distal end of the radius (and ulna)—the so-called childhood Colles' fracture. In middle-aged individuals, especially women, the Colles' type of fracture is the commonest, while Smith's fracture occurs only occasionally.

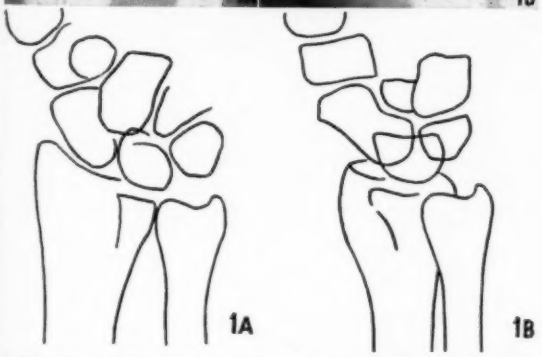
There are several varieties of Colles' and Smith's fractures, ranging from the classical types to the shearing-off of a triangular fragment from the dorsal or volar aspects of the distal end of the radius. In addition, the styloid process of the ulna may be avulsed or it may be fractured without displacement, or the ulnar collateral ligament may be torn or severely stretched.

A new type of fracture (here termed a 'step-fracture') of the distal end of the radius is described below. From a survey of the literature available, it appears that this type of fracture has not been recorded previously.

The mechanism of production is similar to that of a Colles' fracture, viz. a fall on the outstretched hand. At the moment of impact in a Colles' fracture the angle at which the hand comes into contact with the ground is such that the radius undergoes a shearing rotational strain, the shearing occurring in an ulnar and volar direction and the rotation in the direction of pronation. Consequently, the distal fragment, which is displaced dorsally and radially, is also tilted backwards and rotated into relative supination. In the 'step-fracture' the force is transmitted directly in the longitudinal axis of the radius so that the lunate is rammed against the juxtaposed articular surface of the radius with such force that a triangular fragment is displaced proximally. In case 1 illustrated here







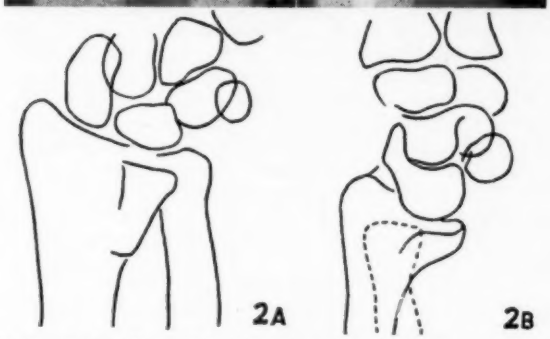


Fig. 1A *and Fig.* 1B. Case 1. X-rays showing step-fracture of distal end of radius; with explanatory line drawings.
Fig. 2A *and Fig.* 2B. Case 2. X-rays showing step-fracture of distal end of radius; with explanatory line drawings. The lunate is out of alignment with the proximal row of the carpus.

the 'step' is a distance of 2 mm. (Figs. 1*a* and 1*b*). In case 2 it is 3 mm. (Fig. 2*a*); furthermore, this fragment is tilted backwards 10—15° (Fig. 2*b*), and it appears that the lunate is out of alignment with the rest of the proximal row of the carpus.

Both patients were middle-aged women who fell on an outstretched hand. In case 1 reduction was attempted, but with no success. Case 2 was seen too late for reduction to be tried. In both instances a Colles'-type plaster cast was applied and retained for 6 weeks. After removal of the plaster, recovery of movements proceeded rapidly and now, 10 and 12 months later respectively, there is a full range of symptom-free movements, with no clinical evidence of instability of the inferior radio-ulnar joint.

SUMMARY

Two cases demonstrating 'step-fractures' of the distal end of the radius are described. As far as we are aware this type of fracture has not been recorded in the literature previously.

We are most grateful to Mr. D. J. Coetzee for the line drawings.

XYLOCAINE INTOXICATION

A REPORT ON THREE RECENT CASES

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Xylocaine (diethyl amino 2-6 dimethyl acetanilide) also known as 'Lidocaine', 'Lignocaine', 'Leostesin' and 'Xylotox', was synthesized by Lofgren and Lunqvist of Sweden in 1943.

Pharmacology. Unlike procaine, xylocaine is quite stable in the presence of acids and alkalis and is not destroyed by boiling. When it is used as an anaesthetic the onset of anaesthesia is rapid, occurring within 2-3 minutes. It can be used for topical as well as infiltration and conduction analgesia. It has no direct vasodilator action and consequently is frequently used without the addition of adrenaline. Its duration of action is approximately 2-5 times greater than that of procaine (Stringer, 1954). A 0·5% solution may produce analgesia for 3-6 hours (Sapeika, 1955).

TOXICITY

Because of its obvious advantages xylocaine has achieved great popularity and is being used with increasing frequency. Like other drugs, however, it produces toxic reactions if used in excessive dosage. Goodman and Gillman (1955) give the toxicity of xylocaine as approximately the same as that of procaine when administered subcutaneously as a 0·5% solution. In a 2% solution they consider it to be 50% more toxic than procaine.

According to Macintosh and Bryce Smith (1953) the toxicity of a local anaesthetic solution increases as the square of the strength of solution used. Hunter (1951) found that in cats, rabbits and rats, the toxicity of xylocaine given intravenously or subcutaneously was twice that of procaine. He states that a 0·5% solution of xylocaine with 1 : 200,000 adrenaline has a potency equal to that of procaine in a 1·0% solution and its duration is very much longer.

The first clinical trials were reported by Gordh (1949). He used the same concentrations of xylocaine as of procaine, but states that because xylocaine is considerably more potent than procaine it should theoretically be possible to use lower concentrations. In 400 cases he observed 2 toxic reactions in adults, both having received a large overdose —1·35 g. and 3·0 g. respectively. No adrenaline was used and he recommends that it should always be added when large amounts of xylocaine are used, the maximum dose of which should not exceed 500 to 1,000 mg.

Bennet (1957) gives a personal account of toxic reaction to an overdose of xylocaine. He developed convulsions without loss of consciousness after 35 c.c. of a 2% xylocaine solution without adrenaline had been infiltrated subcutaneously, i.e. after a total dose of about 600 mg. On 3 previous occasions he had received larger doses but 1 : 400,000 adrenaline had been added to the solutions.

Ansbro *et al.* (1954) in a series of 1,000 cases of caudal anaesthesia, where 2% xylocaine was used, had 11 toxic reactions associated with convulsions. No adrenaline was added to the solutions. They concluded that there was very rapid absorption of the drug by veins in the epidural space.

DeClive-Lowe *et al.* (1954) describe a technique whereby xylocaine and scoline were administered by continuous drip. Only 3 patients out of 400 developed convulsions. From their observations they concluded that it was dangerous to exceed 750 mg. of xylocaine during a procedure lasting 1 hour. The incidence of convulsions would most probably have been much greater had the majority of patients not received 0·5 g. of thiopentone during induction, together with a maintenance infusion of scoline, both drugs having anticonvulsant properties.

Moore (1955) gives the maximum dosage of xylocaine as follows:

Topical: Nose, pharynx and trachea 10 ml. of 2% (200 mg.) urethra 15 ml. of 2% (300 mg.).

Infiltration: 100 ml. of 0·5% (500 mg.), 50 ml. of 1·0% (500 mg.), 25 ml. of 2·0% (500 mg.).

Epidural (lumbar or caudal): 50 c.c. of 1% (500 mg.), 25 c.c. of 2% (500 mg.).

It should be noted that the total dose never exceeds 500 mg.

Adriane and Campbell (1956) have demonstrated that the topical application of local anaesthetics to mucous membranes results in peak blood levels within a few minutes, the rapidity of which closely approaches that of intravenous injection. It is interesting to note that the addition of 1 : 7,500 of adrenaline to their solutions did not retard this rapid absorption from mucosal surfaces.

Toxic Manifestations. The first toxic manifestation is said to be stupor, followed by spasms of the facial muscles, of the upper extremities, and then of the lower extremities together with general depression of respiration and circulation (Dodd, 1954).

Treatment. The first essential in treatment of any systemic reaction to local analgesics should be the administration of oxygen. The initial toxic effects on the cortex and medulla are stimulatory, and may be of such intensity as to produce convulsions. Schmidt (1945) has shown that convulsants may lead to cerebral anoxia (with all its consequences) because they increase the oxygen of the brain beyond the available supply. Respiratory failure resulting from over-stimulation of the respiratory centre, with subsequent depression, should be treated by intermittent positive-pressure artificial respiration, in conjunction with oxygen. Only after adequate oxygenation has been assured should one direct attention to the treatment of convulsions, by minimal intravenous doses of a short-acting barbiturate (Moore, 1955*b*). Larger doses of barbiturate add to the post-stimulatory depression of the medullary centres, resulting in prolonged apnoea. A vasopressor may be indicated if hypotension results from circulatory depression. Cardiac failure is usually secondary to respiratory failure, but may occur primarily, with the rapid entry of large quantities of the drug into the blood stream.

CASE REPORTS

Case 1

A 22-months-old European male child weighing 26 lb. was admitted for an emergency bronchoscopy for the removal of an aspirated peanut. The child was otherwise generally healthy. As some difficulty was anticipated in the removal of the foreign body, it was decided to spray the respiratory tract with a local anaesthetic agent, as a supplement to general anaesthesia. Induction was achieved with nitrous oxide, oxygen, and ether. When the jaw was fully relaxed, the mouth and glottis were sprayed with 4% xylocaine, without adrenaline. A few minutes later, the spray nozzle was directed between the vocal cords, and some of the solution sprayed down the trachea. The total quantity of 4% xylocaine used was 2 ml. (80 mg.). Approximately 2 minutes after spraying, twitches of the eyelids occurred. This was soon followed by facial twitches, and then generalized convulsions. Oxygen by mask was immediately administered, and the convulsions were controlled by the intravenous injection of 3 ml. of 2½% thiopentone. The operation was concluded without further mishap. Circulation and respiration were not adversely affected.

Discussion. The total maximum adult dose for topical application to the nasopharynx and trachea is given as 200 mg. (Moore, 1955*a*). Assuming the average adult weight to be 154 lb. (70 kg.), the dose of xylocaine per lb. body-weight will be about 0·8 mg. On this basis the maximum dose for a child weighing 26 lb. would be approximately 21 mg., so that the dose administered in this case (80 mg.) was almost 4 times the maximum dose, and the concentration used was double the recommended 2%. According to Lee (1953) ether increases the susceptibility to the toxic effects of local analgesics, so that it may have been a contributory factor in this case, but overdosage is undoubtedly the main factor.

Case 2

A European male child weighing 28 lb., aged 24 months, was admitted suffering from an acute laryngo-tracheo-bronchitis. With the onset of severe laryngeal stridor and dyspnoea, an emergency tracheotomy was considered necessary under local infiltration anaesthesia. The operative site was infiltrated with 7 ml. of 2% xylocaine (140 mg.) without adrenaline.

This was followed approximately 8 minutes later, after the tracheotomy was completed, by facial twitches, clonic contractions of the limbs, and then generalized convulsions. Intravenous injection of 3 ml. of 2½% thiopentone was necessary to relieve the convulsions. Spontaneous respiration did not return until approximately 10 minutes later. During this period intermittent positive-pressure artificial respiration was performed *via* an endotracheal tube inserted into the tracheostome, through which oxygen together with a small percentage of carbon dioxide was delivered. There was no apparent circulatory disturbance and complete recovery resulted.

Discussion. According to Moore (1955*a*) the total maximum adult dose for infiltration is 500 mg. This represents approximately 3 mg. per lb. body-weight, and the total maximum dose for a child weighing 28 lb. is approximately 84 mg. The dose administered was 140 mg. so that this must also be considered a case of overdosage.

Case 3

A Coloured primipara aged 23 years, weighing approximately 125 lb., sustained a third-degree tear during delivery of an infant. It was decided to repair the tear under a pudendal nerve block. 10 ml. of a 2% solution of xylocaine was infiltrated transvaginally on either side, the procedure taking about 7 minutes to complete. After a wait of about 5 minutes, the analgesia was not considered satisfactory; so a further 10 ml. of a 2% xylocaine was used to infiltrate the tissues around the tear. While this was being done the patient developed twitches of the hands and feet, but this was thought to be due to nervousness and repair of the perineal tear commenced. It was then noticed that the patient was unconscious and was having generalized twitches. This was 4 minutes after the last 10 ml. of xylocaine had been infiltrated. Generalized convulsions followed. The pulse at this stage was 120 beats per minute and of good volume. Oxygen was administered from a Boyle's-type anaesthetic machine and 50 mg., of 2½% thiopentone was given intravenously, which stopped the convulsions within 1 minute; 15 minutes later the patient regained consciousness. The systolic blood pressure was found to be 120 mm. Hg. Repair of the tear was completed and no further toxic manifestations occurred.

Discussion. This patient received a total of 30 ml. of 2% xylocaine without adrenaline. This represents 600 mg. administered within a short period into a very vascular region, so that absorption must have occurred rapidly, and was probably the main factor in producing a toxic reaction.

CONCLUSION

It would appear that most toxic reactions following the use of xylocaine are due to overdosage, resulting from failure to realize its greater potency as compared with procaine, and from the tendency to omit the addition of adrenaline to solutions. The total maximum dose should not be exceeded, and in most instances regional blocks may be executed effectively with amounts far less than the maximum dose, provided the procedure is conducted with reasonable skill. The weakest concentration which will produce the desired analgesia should be used, and in all instances adrenaline should be added unless specifically contra-indicated.

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COLLEGE OF PHYSICIANS AND SURGEONS OF SOUTH AFRICA

Minutes have been circulated of the first annual general meeting of the College which was held on 6 August 1956 at Medical House, Esselen Street, Johannesburg. Dr. A. W. S. Sichel, Chairman of the Steering Committee, presided and Mr. T. B. McMurray, Hon. Secretary and Treasurer of the Steering Committee, was in attendance.

The Chairman stated that the Steering Committee had continued in office since it was appointed in 1954 and had effected the alterations in the Constitution recommended by the Inaugural Meeting. The College had now been registered as a non-profit-making Company and the first Council had been elected. The Steering Committee was to vacate office and hand over to the first Council. A vote of thanks was passed expressing sincere gratitude to the Steering Committee.

Reference was made to the loss of two Founders, viz., Mr. L. B. Goldschmidt, who had bequeathed £500 to the College to be devoted to some aspect of Urology, and Mr. M. Cole Rous, who had been Vice-chairman of the Steering Committee.

It was agreed to recommend to the Council to apply to the College of Heralds, London, for a College coat of arms, and also to obtain a suitable gown for the President.

A resolution was passed concerning the formation of Divisions and Faculties in the College. The meeting further authorized the formation of further Faculties at the discretion of the Council.

The question of the desirability of changing the name of the College was referred to the Council and will come up again at the next annual general meeting. The President of the College has prepared a memorandum on the subject.

Amendments were passed to the Constitution concerning the voting rights of Founders, Associate Founders and Fellows, the powers of proxy and the discussion rights of Members. (The relative clauses in the Constitution were approved at an extraordinary general meeting of the College held on 22 November 1956 at the College office, Cape Town.)

A motion to form a separate Division of Obstetrics and Gynaecology within the College was passed *nem. con.* (The consequential alterations in the Articles of Association were approved at the extraordinary general meeting of 22 November 1956.)

On a motion that the headquarters of the College should be in Johannesburg, an amendment was passed referring the matter to the Council. (The Council has subsequently decided that no good purpose would be served in laying down a site at present.)

It was decided to found a Journal for the College. (The Council has decided that in the first instance the *Transactions of the College of Physicians and Surgeons* would be published under the editorship of Mr. A. J. Helfet, the Hon. Librarian.

The Election of the first Council was reported as follows: Prof. G. A. Elliott, Dr. S. C. Heyman, Mr. W. H. D. Trubshaw, Mr. J. A. Douglas, Dr. M. M. Suzman, Mr. A. J. Helfet, Mr. T. B. McMurray, Dr. A. W. S. Sichel, Prof. J. F. Brock, Prof. F. Forman, Mr. A. G. Sweetapple, Dr. R. Theron.

Next Annual General Meeting

The second annual general meeting of the College of Physicians and Surgeons of South Africa will be held at Red Cross House, Durban, on 14 September 1957 at 10.30 a.m.

OFFICIAL ANNOUNCEMENT: AMPTELIKE AANKONDIGING

APPROVED MEDICAL AID SOCIETIES: GOEDGEKEURDE MEDIESE HULPVERENIGINGS

MEDICAL AID SOCIETIES

The following list of approved medical aid societies is published for general information. Members are requested to keep this list for reference because it will no longer appear in the tariff book. After each meeting of the Federal Council an up-to-date list will be published in the *Journal*, including societies that have been newly approved and omitting those that have been withdrawn.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
20 August 1957

1. A.A. Mutual Medical Aid Society, P.O. Box 9595, Johannesburg.
2. Abercom Group Sick Benefit Society, P.O. Box 715, Port Elizabeth, Cape Province.
3. African Cables Medical Benefit Fund, P.O. Box 172, Vereeniging, Transvaal.
4. African Explosives Medical Aid Society, P.O. Box 1122, Johannesburg.
5. African Homes Trust Sick Fund, P.O. Box 93, Cape Town.
6. African Oxygen & Acetylene Medical Aid Society, P.O. Box 5404, Johannesburg.

MEDIESE HULPVERENIGINGS

Vir algemene inligting word onderstaande lys van goedgekeurde mediese hulpverenigings gepubliseer. Lede word versoek om die lys byderhand te hou want in die vervolg word dit nie in die tariefboek geplaas nie. Na elke vergadering van die Federale Raad sal 'n volledige lys (wat die name van pas-goedgekeurde verenigings insluit en van dié wat onttrek is weglaat) in die *Tydskrif* verskyn.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
20 Augustus 1957

7. Afrikaanse Pers Beperk se Siekefonds, Posbus 845, Johannesburg.
8. Alex. Aiken & Carter Medical Benefit Society, P.O. Box 2636, Johannesburg.
9. Algoa Medical Aid Society, P.O. Box 369, Port Elizabeth.
10. Anglo-Alpha (Dudfield) Medical Benefit Society, Private Bag, P.O. Lichtenburg, Transvaal.
11. Argus Medical Benefit Society (Cape Argus Branch), P.O. Box 56, Cape Town.
12. Argus Medical Benefit Society (Daily News Branch), P.O. Box 1491, Durban.

13. Argus Medical Benefit Society (Star Branch), P.O. Box 1014, Johannesburg.
14. A.T.I. Medical Aid Society, P.O. Box 5057, Boksburg North.
15. Atlantic Refining Company Medical Aid Society, P.O. Box 664, Cape Town.
16. Babcock and Wilcox Medical Aid Fund, P.O. Box 545, Vereeniging.
17. Bakers Ltd. European Employees' Sick Benefit Fund, P.O. Box 692, Durban.
18. Bloemfontein Municipal Employees' Medical Aid Society, P.O. Box 288, Bloemfontein.
19. Boksburg Municipal Employees' Medical Aid Fund, P.O. Box 215, Boksburg.
20. Broderick Medical Aid Society, P.O. Box 186, Vereeniging.
21. Building Societies Joint Medical Aid Fund, P.O. Box 5728, Johannesburg.
22. S. Butcher & Sons Ltd. Medical Aid Society, P.O. Box 1004, Durban.
23. Caltex Medical Aid Society (S.A.), P.O. Box 714, Cape Town.
24. Cape Times Medical Aid Society, P.O. Box 11, Cape Town.
25. Cape Town Municipal Employees' Association Medical Aid Society, P.O. Box 1939, Cape Town.
26. Central News Agency Ltd. Medical Benefit Society, P.O. Box 1033, Johannesburg (excluding Cape Town and suburbs, Durban municipal area, Johannesburg and Witwatersrand, and Port Elizabeth and Pretoria municipal areas).
27. Chamber of Mines Medical Aid Society, P.O. Box 809, Johannesburg.
28. Civil Service Medical Benefit Association, P.O. Box 176, Pretoria.
29. Consolidated Glassworks Limited Medical Aid and Sick Benefit Society, P.O. Box 562, Germiston.
30. Corner House Insurance Fund, P.O. Box 1056, Johannesburg.
31. Coronation Medical Aid Society, P.O. Box 1517, Durban.
32. Crookes Bros. Ltd. Medical Benefit Fund, 301 Smith Street, Durban.
33. D.F.A. Medical Benefit Society, P.O. Box 610, Kimberley.
34. Eastern Province Cement Co. Ltd. Medical Aid Society, P.O. Box 2016, Port Elizabeth.
35. E.P. Newspapers Medical Aid Society, P.O. Box 1117, Port Elizabeth.
36. Egnep Medical Aid Society, P.O. Penge, Transvaal.
37. Elwamba Medical Aid Fund, P.O. Box 42, East London.
38. Escom (N.C.U.) Medical Benefit Society, P.O. Box 30, Colenso, Natal.
39. Everite Medical Aid Society, P.O. Kliprivier, Transvaal.
40. Federated Employers' Medical Aid Society, P.O. Box 666, Johannesburg.
41. Federation of Master Printers of S.A. Medical Aid Society, P.O. Box 1200, Johannesburg.
42. Ford Medical Aid Society, P.O. Box 788, Port Elizabeth.
43. Friend Medical Aid Fund, P.O. Box 245, Bloemfontein.
44. General Mining (Associated Companies) Medical Aid Society, P.O. Box 29, Luipaardsvlei, Transvaal.
45. General Motors Medical Aid Scheme, P.O. Box 1137, Port Elizabeth.
46. Germiston Industries Medical Aid Society, 113 Pylon House, Human Street, Germiston.
47. Gledhow-Chaka's Kraal Sugar Co. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
48. Greaterman's Medical Aid Society (all Branches), P.O. Box 5460, Johannesburg.
49. Hollerith Medical Aid Society, P.O. Box 7018, Johannesburg.
50. Hubert Davies Johannesburg Staff Medical Aid Society, P.O. Box 1386, Johannesburg.
51. Sir J. L. Hulett & Sons Ltd. Medical Benefit Fund, P.O. Box 248, Durban.
52. Hume Cape Medical Benefit Society, P.O. Box 7, Bellville, Cape Province.
53. Hume Transvaal Medical Benefit Society, P.O. Box 204, Germiston.
54. Hunt, Leuchars & Hepburn Ltd. (Durban) Employees' Medical Benefit Fund, P.O. Box 943, Durban.
55. Hunt, Leuchars & Hepburn Ltd. (Transvaal Staff) Medical Aid Society, P.O. Box 47, Johannesburg.
56. Iscor Medical Benefit Fund, P.O. Box 450, Pretoria.
57. I.W.S. Medical Aid Society, P.O. Box 6946, Johannesburg.
58. J. W. Jagger & Co. Ltd. Medical Aid Society, P.O. Box 726, Cape Town.
59. Johannesburg Board of Executors' Medical Aid Society, P.O. Box 271, Johannesburg.
60. Klerksdorp Munisipale Werknemers Siektefonds, Posbus 99, Klerksdorp.
61. K. & L. Timbers Ltd. Staff Medical Aid Fund, P.O. Box 9, Elandsfontein, Transvaal.
62. Koegas Medical Aid Society, P.O. Koegasbridge, C.P.
63. Krantzberg Mines Medical Aid Society, P.O. Box 18, Omaruru, S.W.A.
64. Kroonstad Munisipale Mediese Hulpvereniging, Posbus 302, Kroonstad.
65. G. H. Langler & Co. Ltd. Medical Aid Society, P.O. Box 3762, Johannesburg.
66. Legal and General Medical Aid Society, P.O. Box 4870, Johannesburg.
67. Mail Times & Express Medical Aid Society, P.O. Box 1138, Johannesburg.
68. L. H. Marthinusen Medical Aid Society, P.O. Box 64, Denver, Johannesburg.
69. Masonite Medical Aid Society, P.O. Box 57, Estcourt, Natal.
70. Metal Box Company of S.A. Ltd. Medical Aid Society, P.O. Box 7752, Johannesburg.
71. Municipal Employees' Medical Aid Society (Durban), P.O. Box 625, Durban).
72. Natal Building Society Medical Aid Fund, P.O. Box 947, Durban.
73. Natal Coal Owners' (Durban Staff) Medical Aid Society, P.O. Box 281, Durban.
74. Natal Estates Sick Fund Benefit Society, P.O. Mount Edgecombe, Natal.
75. Natal Industries Medical Aid Society, P.O. Box 1300, Durban.
76. N.T.E. Staff Medical Aid Fund, P.O. Box 39, Pietermaritzburg.
77. National Industrial Credit Corporation Medical Aid Society, P.O. Box 8296, Johannesburg.
78. National Portland Medical Aid Society, P.O. Box 21, Claremont, C.P.
79. New Consolidated Gold Fields Employees' Medical Aid Fund, P.O. Box 1167, Johannesburg.
80. Northern Medical Aid Society, P.O. Box 3437, Johannesburg.
81. Northern Rhodesia Civil Servants Medical Aid Society, P.O. Box 294, Lusaka, Northern Rhodesia.
82. Norwich Union Life Insurance Society Staff Medical and Surgical Benefit Scheme, P.O. Box 1226, Cape Town.
83. Ore & Metal Medical Aid Society, P.O. Box 3548, Johannesburg.
84. Pietermaritzburg Chamber of Industries Medical Aid Society, P.O. Box 365, Pietermaritzburg.
85. Polliack Group Medical Aid Society, P.O. Box 3008, Johannesburg.
86. Pongola Sugar Milling Co. Ltd. Medical Benefit Fund, P.O. Box 194, Durban.
87. Post Office Medical Aid Society, P.O. Box 303, Germiston.
88. Pretoria Municipal Employees Sick Fund, P.O. Box 408, Pretoria.
89. Pretoria News Medical Benefit Society, P.O. Box 439, Pretoria.
90. Pretoria Portland Cement Co. Ltd. No. 1 Works (Hercules) Medical Aid Society, P.O. Box 405, Pretoria.
91. Pretoria Portland Cement Co. Ltd. No. 2 Works Medical Benefit Society, P.O. Box 7, Slurry, Western Transvaal.
92. Pretoria Portland Cement Co. Ltd. No. 3 Works (Jupiter) Medical Aid Society, P.O. Box 73, Cleveland, Transvaal.
93. Pretoria Portland Cement Co. Ltd. No. 4 Works Medical Aid Society, P.O. Box 26, Orkney, District Klerksdorp.
94. Printing Industry Medical Aid Society, P.O. Box 1993, Pretoria.
95. Prudential Medical Aid Scheme, P.O. Box 1097, Cape Town.

96. Rand Public Service Medical Aid Society, P.O. Box 28, Boksburg.
97. Rand Water Board Sick Fund, P.O. Box 1127, Johannesburg.
98. Randles Bros. & Hudson Ltd. (Durban) Sick Benefit Fund, P.O. Box 1046, Durban.
99. Randles Bros. & Hudson Ltd. (Johannesburg) Employees' Sick Benefit Fund, P.O. Box 2678, Johannesburg.
100. Reckitt & Colman Medical Aid Society (S.A.), P.O. Box 1097, Cape Town.
101. 'Rennie' and 'The Consolidated' Employees' Medical Aid Fund, P.O. Box 1006, Durban.
102. Reunert & Lenz Ltd. Medical Aid Society (All Branches), P.O. Box 92, Johannesburg.
103. Reynolds Bros. Ltd. Medical Benefits Fund, 301 Smith Street, Durban.
104. E. S. & A. Robinson (Pty.) Ltd. Medical Aid Society, P.O. Box 293, Germiston.
105. Royal-Globe Medical Aid Fund, P.O. Box 317, Cape Town.
106. Safim Medical Aid Society, P.O. Box 233, Vereeniging.
107. Safmarine Medical Aid Society, P.O. Box 2171, Cape Town.
108. Safnit Mills Medical Aid Fund, P.O. Box 11, Jeppestown, Johannesburg.
109. Santam-Sanlam Siektefonds (Alle Takke), Posbus 1, Sanlamhof, K.P.
110. Schwartz, Fine, Kane & Co. Medical Aid Society, P.O. Box 5069, Johannesburg.
111. Shell Medical Aid Society (S.A.), P.O. Box 2231, Cape Town.
112. Siektefonds van Wolgroeiers Afslaers Beperk, Posbus 765, Port Elizabeth.
113. C. G. Smith & Co. Ltd. Medical Aid Fund, 301 Smith Street, Durban.
114. S.A. Association of Municipal Employees' (S.A.A.M.E.) Medical Aid Fund, P.O. Box 62, Pretoria.
115. S.A. Breweries Medical Aid Society, P.O. Box 1099, Johannesburg.
116. S.A.K.A.V. Sick Benefit Fund, P.O. Box 33, Paarl.
117. S.A. Mutual Fire & General Insurance Co. Ltd. Staff Medical Aid Fund, P.O. Box 516, Johannesburg.
118. S.A. Mutual Life Assurance Society Staff Medical Aid Fund, P.O. Box 66, Cape Town.
119. S.A. Press Association Medical Aid Society, P.O. Box 7766, Johannesburg.
120. S.A. Teachers' Association Medical Aid Society, 12 Bellevue Road, Sea Point, C.P.
121. S.A. Torbanite (Boksburg) Medical Aid Society, P.O. Box 5038, Boksburg North.
122. South Atlantic Corporation Medical Aid Society, P.O. Box 4610, Cape Town.
123. Southern Medical Aid Society, P.O. Box 42, Cape Town.
124. Standard Brass Medical Aid Society, P.O. Box 229, Benoni.
125. Stewarts & Lloyds Medical Benefit Fund, P.O. Box 74, Vereeniging.
126. Stuttafords Medical Aid Society, P.O. Box 69, Cape Town.
127. Sun Insurance Office Ltd. Staff Medical Aid Fund, P.O. Box 429, Johannesburg.
128. Syfret's Medical Aid Society, 24 Wale Street, Cape Town.
129. Traduna Medical Aid Fund, P.O. Box 8791, Johannesburg.
130. Transvaal Corundum Associated Asbestos Medical Aid Society, P.O. Box 72, Pietersburg, Transvaal.
131. Transvaal Society of Accountants Medical Aid Fund, P.O. Box 2995, Johannesburg.
132. U.L.A. Medical Aid Society, P.O. Box 4589, Johannesburg.
133. Umzimkulu Sugar Co. Ltd. Medical Aid Fund, P.O. Box 43, Durban.
134. United Banks' Medical Aid Society, P.O. Box 1242, Cape Town.
135. United Building Society Medical Benefit Fund, P.O. Box 7735, Johannesburg.
136. University of the Witwatersrand (Johannesburg) Staff Medical Aid Fund, Milner Park, Johannesburg.
137. Vacuum Medical Aid Society, P.O. Box 35, Cape Town.
138. Village Board of Management of Welkom Medical Aid Society, P.O. Box 708, Welkom, O.F.S.
139. Wright Boag & Head Wrightson Sick Benefit Fund, P.O. Box 183, Benoni.
140. Yorkshire Medical Aid Society, P.O. Box 2755, Johannesburg.

MEDICAL BENEFIT SOCIETIES WHICH ALLOW FREE CHOICE OF DOCTOR FOR SPECIALIST SERVICES ONLY: MEDIESE BYSTANDSVERENIGINGS WAT VRY KEUSE VAN DOKTER ALLEEN VIR SPESIALISTEDIENSTE TOELAAT

1. Begbie Medical Benefit Fund, P.O. Box 192, Middelburg, Transvaal.
2. Breyten Coalfields Benefit Society, P.O. Box 6, Estantia, Transvaal.
3. De Beers Consolidated Mines Limited Benefit Society, P.O. Box 616, Kimberley.
4. Durban Roodepoort Deep Ltd. Benefit Society, P.O. Box 193, Roodepoort.
5. Jagersfontein Mine Benefit Society, P.O. Box 2, Jagersfontein, O.F.S.
6. Krugersdorp Municipal Employees' Medical Benefit Society, P.O. Box 101, Krugersdorp.
7. Northern Rhodesia Mine Employees Medical Specialist Fund, P.O. Box 134, Kitwe, Northern Rhodesia.
8. Public Utility Transport Corporation Sick Fund, P.O. Box 9571, Johannesburg.
9. Randfontein Estates Employees' Sick Benefit Society, P.O. Box 37, Randfontein.
10. Roodepoort-Maraisburg Municipal Employees' Association Sick Benefit Society, P.O. Box 217, Roodepoort.
11. Roodepoort-Maraisburg Non-Scheduled Mines' and Industries' Benefit Society, P.O. Box 225, Roodepoort.
12. Rosherville-Maraisburg Benefit Society, P.O. Box 99, Cleveland, Johannesburg.
13. Sasol Medical Benefit Society, P.O. Box 80, Sasolburg.
14. Simmer Pan Medical Benefit Society, P.O. Box 103, Germiston.
15. Springs Mines Benefit Society, P.O. Box 54, Springs.
16. Transvaal Jewellers' & Goldsmiths' Sick Benefit Fund, P.O. Box 8530, Johannesburg.
17. Witbank Coalfields Benefit Society, P.O. Box 26, Witbank.
18. Witbank Power Station Medical Benefit Society, P.O. Box 197, Witbank.

FRANK FORMAN MEDICAL FOUNDATION : 1958 AWARD

The Board of Trustees of the Frank Forman Medical Foundation announce that:

1. The sum of seven hundred pounds (£700) will be available for postgraduate award for the year 1958.
2. In terms of the Trust Deed, the Board of Trustees are directed to use their discretion in making the award, in such manner as shall promote or assist the study of medicine and/or medical research at the University of Cape Town or elsewhere.
3. The award may take the form of a *Scholarship* to a medical graduate for postgraduate medical study for 1 year; and/or

A *Fellowship* to a university graduate for postgraduate medical research for 1 year; and/or

A *Grant*, either independently or in conjunction with other research grants, to any person or institution or body for special medical investigation or research.

4. Applications should be addressed to the Secretary, Frank Forman Medical Foundation, P.O. Box 46, Cape Town, and must reach him before 1 October 1957.

AMENDMENTS TO WORKMEN'S COMPENSATION ACT TARIFF

Under Section 79 of the Workmen's Compensation Act No. 30 of 1941, the Workmen's Compensation Commissioner gives notice in Government Notice No. 686 of 16 August 1957 that, after consultation with the Medical Association of South Africa, he has approved of the corrections and amplifications of the scale of fees published in Government Notice No. 1668 of 1956, with effect from 1 May 1957, as set out as follows:

Natives							*Non-Natives*					
General Practitioner			*Specialist*				*General Practitioner*			*Specialist*		
£	s.	d.	£	s.	d.		£	s.	d.	£	s.	d.
						1. *Item* 5						
						Insert new sub-item (12):						
3	5	0	5	0	0	Suture of muscle or tendon not elsewhere provided for	5	0	0	8	0	0
						2. *Item* 7						
						In sub-item (1) delete the words 'and foreign bodies in back of hand'.						
						In sub-item (3) delete the words 'palm of' and insert the words 'under general anaesthetic' after the word 'foot'.						
						3. *Item* 11						
						In sub-item (2) (*k*) insert the words 'or Discography' after the word 'Myelography'.						
						In sub-item (3) (*j*) delete the fees '£18, £27, £27, £40' and the word 'Major' and substitute the following therefor:						
13	5	0	20	0	0	With removal/elevation of bone	20	0	0	30	0	0
						Delete the existing sub-items (3) (*k*) and substitute therefor new sub-item (3) (*k*):						
						Repair of compound fracture of skull with dural involvement:						
20	0	0	30	0	0	Without brain laceration	30	0	0	45	0	0
35	5	0	53	0	0	With brain laceration	53	0	0	80	0	0
						Delete the existing sub-item (7) (*b*) and substitute therefor new sub-item (7) (*b*):						
						Fees for assistant at neuro-surgical operation (excluding investigations):						
						£5 for the first hour and £1 5s. 0d. for every 15 minutes or portion thereof, thereafter, with a maximum of £15.*						
						4. *Item* 21						
						Delete the existing sub-item (3) (*a*) and substitute therefor new sub-item (3) (*a*):						
						Traumatic Cataract						
13	5	0	20	0	0	(i) Needlings (inclusive fee)	20	0	0	30	0	0
18	0	0	27	0	0	(ii) Needling followed by Curette Evacuations	27	0	0	40	0	0
22	0	0	33	0	0	(iii) Intra-capsular Extraction	33	0	0	50	0	0
22	0	0	33	0	0	(iv) Extra-capsular Extraction including subsequent needlings.. ..	33	0	0	50	0	0
						In sub-item (5) delete the fee '£17 10s. 0d.' laid down for specialists and substitute '£18 0s. 0d.' therefor						
						Insert new sub-items:						
18	0	0	27	0	0	(19) Dacryocystotomy	27	0	0	40	0	0
0	10	0				(20) Orthoptic treatment—per treatment	0	12	6			
0	15	0	1	10	0	(21) Hess Chart	1	0	0	2	0	0
						The fees prescribed for General Practitioners in sub-items (20) and (21) above, shall also apply to Orthoptists.						
						5. *Item* 23						
						After sub-item (1) (*a*) insert the following explanatory note:						
						Where more than two fingers or toes are X-rayed at the same time the maximum fee would be that laid down under sub-item (1) (*b*).						
						In sub-item (17) (*b*) delete the words 'or deep' and insert new sub-item (iii):						
						Deep X-ray Therapy per treatment						
			1	0	0	One field or area				1	10	0
			1	0	0	Additional fields or lesions each				1	10	0
						Note In no case shall more than 20 treatments (all fields or areas or lesions included) be paid for unless the prior approval of the Commissioner has been obtained.						
						In sub-item (17) (*c*) insert new sub-item (iii):						
			17	0	0	Operative insertion of Radium, Radon, or other Radio-active substance.				25	0	0
						6. *Item* 29						
						In sub-item (1) (*a*) delete the fees laid down for Natives and substitute the following therefor:						
0	10	0	0	15	0							

* The Workmen's Compensation Commissioner rules that this fee will only apply to assistants who are registered specialists. General practitioner assistants will receive the usual 10% of the surgical fee.

IN MEMORIAM

H. Q. FORSYTH THOMPSON M.R.C.S., (ENG.) L.R.C.P. (LOND.),

Humphrey Quentin Forsyth Thompson, born in 1891 at Springvale near Ixopo, Natal, was educated at his father's school, Weenen County College, in Natal, and later received his medical training at Guy's Hospital, London. There he was awarded the Hilton prize for surgery, and he got his blue for Rugby.

On qualifying in 1915 he was appointed to the King Edward VII Hospital for officers, and after a brief spell there joined what was called the Anglo-Russian Field Hospital, which Lady Muriel Paget was largely responsible for raising. He served on various fronts in Russia, but particularly in the Carpathians where, in two battles, he was the only medical officer in charge of the field hospital. He was awarded the (Russian) Cross of St. George for bravery and devotion to duty, and also decorations in the orders of St. Vladimir and St. Stanislas.

Dr. H. Q. Forsyth Thompson

On the replacement of the Tsarist regime by the Bolshevik in 1917, the Anglo-Russian Field Hospital was dispersed and evacuated with great difficulty. Dr. Forsyth Thompson escaped to Japan with greatly impaired health. There he recuperated for a month or so, and then found his way back to South Africa.

After doing some locums in Durban and a brief spell in the Johannesburg General Hospital, he was engaged by the Corner House group of mines (in 1919) and held appointments as Medical Officer successively in the City Deep, Modder B and Crown Mines.

He was one of those fortunate people who 'learnt languages easily'. He spoke several European languages, including Russian; but it was his fluency in Zulu (which he learnt as a boy), Xosa and Sutho that so facilitated his work on the mines. He was also completely at home in Afrikaans.

During the second World War he acted as Senior Medical Officer of the Group. At the end of the war and on the return of General Orenstein, he again became the Medical Officer of the Crown Mines.

During his latter years on the Rand he had heart trouble, as a result of which he resigned at the end of 1950 and came to live in the Cape.

After some years of irksome inactivity, he decided to have his name restored to the Medical Register and try and do a little work. It was while he was relieving a medical officer at one of the Peninsula clinics that he caught a chill which developed into pneumonia from which he died on Wednesday, 14 August 1957.

He is survived by his widow, who was the second daughter of the late Col. R. W. and Mrs. Studdy, of Waddeton Court, Brixham, Devonshire.

Dr. L. E. Miller, of Roodepoort, Transvaal, writes: Humphrey Quentin Forsyth Thompson was born in Natal on 30 January 1891 and died at Kenilworth, C.P. on 14 August 1957. He did his medical course at Guy's Hospital, London, taking the M.R.C.S. (Eng.), L.R.C.P. (Lond.) in January 1915.

He proceeded to Russia with the Russian Expeditionary Force in 1915 and remained in Russia for 3 years, where he rendered distinguished service, attaining the rank of Colonel in the Russian Army. He was captured by the Bolsheviks in 1918, and together with other British prisoners was sentenced to death. However, he managed to escape *via* Japan and eventually returned to Britain.

In 1919 he came to Johannesburg and took up an appointment at the Johannesburg General Hospital as houseman. In January 1921 he joined the medical services of the Rand Mines Ltd. and was with the group until his retirement on account of ill health in December 1950. He was an active member of the Transvaal Mine Medical Officers' Association and was elected President in 1931. He was the author of publications on Tropical Ulcers, Knee-Joint Injuries, Pneumonia, Bantu tribal amputations of parts of fingers, etc., etc.

During World War II he acted as Chief Medical Officer for the Rand Mines Group during the absence on active service of Major-General A. J. Orenstein, and whilst holding this office was chairman of a committee which, under the auspices of the South African Red Cross Society, published the *South African Digest of War Medicine*.

He was an outstanding linguist and it was a pleasure to hear him converse with the Bantu, no matter whether he was a Zulu, a Basuto or a Xosa. After his sojourn in Russia, he spoke Russian fluently. Having no knowledge of Afrikaans, he learnt it in a year while at the General Hospital and obtained a first-class pass in the Taalbond examination.

Humphrey Thompson was a man of great personal charm and kindliness; one took a liking to him instantly. His passing will be mourned by a wide circle of friends and colleagues and by as wide a circle of Africans, with whom he came into close contact in his work on the Mines and who held him in the highest esteem.

ASSOCIATION NEWS : VERENIGINGSNUUS

KENNISGEWING

Die Jaarvergadering van die Distriksgeneeshere-Vereniging van Suid-Afrika, sal gehou word tydens die Mediese Kongres op Vrydag, 20 September 1957 om 2.15 nm. in die Rooi Kruis-gebou, Old Fort-weg, Durban.

1. Lees van Kennisgewing van vergadering.
2. Notule van vorige algemene vergadering.
3. Voorsitter se jaarverslag.
4. Finansiële verslag.
5. Toespraak deur Sekretaris van Volksgesondheid.
6. Verkiesing van nuwe bestuur.
7. Algemeen.

PASSING EVENTS : IN DIE VERBYGAAN

Federal Council Elections. The following further results of the recent 5-yearly Branch Elections for Federal Council have been received:

Border, R. Schaffer, L. L. Alexander, J. K. McCabe.
Griqualand West, N. Kretzmar.

Dr. Pincus Catzel, M.B., B.Ch. (Rand), M.R.C.P. (Edin.), D.C.H., R.C.P. & S. (Eng.), has joined Dr. C. van Waalwijk van Doorn in his practice as Specialist in Diseases of Children at 401 Medical Centre, Pretorius Street, Pretoria. Telephone: rooms 2-0302, residence 4-3284.

Mr. F. P. Jacobsz, F.R.C.S. (Edin.), previously of Bloemfontein, has started in practice as a specialist surgeon at 211 Sanlam Building, Smith Street, Durban. Telephones: consulting rooms 21575, after hours 29326.

* * *

South African Paediatric Association. The next meeting of the Cape Town Sub-group of this Association will be held on Tuesday 3 September 1957 in the Lecture Theatre, Red Cross War Memorial Children's Hospital, Rondebosch, Cape, at 8.15 p.m. Dr. Walter Phillips will address the meeting on 'New Methods in Heart Surgery.

* * *

Dr. M. Cecil Michelow, M.B., B.Ch. (Rand), D.Obst., R.C.O.G., M.R.C.O.G., has commenced practice as an Obstetrician and Gynaecologist in partnership with Drs. S. Joel Cohen and A. M. Porter at 208 Medical Centre, Jeppe Street, Johannesburg. Telephones: consulting rooms 23-7124 and 42-1218, residence 41-4105, emergency 22-4191 (these numbers are not in the current telephone directory).

* * *

The Organizing Committee of the South African Medical Congress, to be held in Durban on 16-21 September 1957 suggests to members that they would be saved much inconvenience, time and trouble in registration if they would register and pay for all tickets by post. From the intention cards it appears that tickets both for the banquet and the ball will be at a premium, but so far few members have actually paid for the tickets.

* * *

Dr. D. M. T. Gairdner, Consultant Paediatrician, Addenbrooke's Hospital, Cambridge, England, who will visit South Africa to attend the Medical Congress at Durban, will address the Cape Midland Branch at Port Elizabeth on Monday, 23 September, the Cape Western Branch at Cape Town on Wednesday, 25 September (on the Henoch-Schönlein syndrome), and a combined Paediatric Group meeting at Johannesburg on Wednesday, 2 October.

* * *

The Wellcome Trustees have made a grant of up to £80,000 to the Government of Kenya towards the cost of building and equipping a special establishment for research on foot-and-mouth disease in that country. The Institute, which will be sited in the Nairobi area, will be under the control of the Kenya Veterinary Department and will be in the charge of Mr. J. W. Macaulay, F.R.C.V.S., B.Sc., D.V.S.M. It will be known as 'The Wellcome Institute for Research on Foot and Mouth Disease'.

* * *

Seventh International Cancer Congress. Those planning to attend the 7th International Cancer Congress, which will take place at the Royal Festival Hall, London, on 6-12 July 1958, are reminded that enrolment forms must be received at the Congress Office (45 Lincoln's Inn Fields, London, W.C.2) *by 1 January* 1958 if a late fee is not to be incurred. Registration forms and the Preliminary Programme may be obtained from the Secretary-General at that address.

* * *

Union of South Africa. Department of Health. Notification of formidable epidemic diseases and poliomyelitis in the Union during the period 9-15 August 1957.

	Poliomyelitis				
	Eur.	*Nat.*	*Col.*	*As.*	*Total*
Transvaal	–	1	–	–	1
Cape Province ..	2	–	1	–	3
Orange Free State ..	–	1	–	–	1
Natal	–	3	–	–	3
Totals	2	5	1	–	8

Plague, Smallpox, Typhus Fever: Nil.

* * *

The Silver Jubilee Meeting of the Association of Anaesthetists of Great Britain and Ireland will take place on 4-7 December 1957 at the Royal College of Surgeons, Lincoln's Inn Fields, London, W.C.2. A symposium on anaesthetic apparatus and equipment and a panel discussion on anaesthetic apparatus will be held as well as other scientific sessions. Dr. M. D. Nosworthy will deliver the Frederic Hewitt Lecture, and Dr. B. L. S. Murtagh the presidential address of the Section of Anaesthetists of the Royal Society of Medicine. A number of firms will be exhibiting throughout the meeting, and there will be a small scientific exhibition.

The overseas visitors are invited to a cocktail party by invitation of the President and Council.

The registration fee is £2 0s. 0d. including morning coffee, lunches and teas. The annual dinner will be held in the Great Hall, Royal College of Surgeons, and a buffet dance at Londonderry House, Park Lane, London, W.1, for which tickets may be bought.

* * *

The South African Society of Anaesthetists. The Cape Western Sub-group of this Society has arranged a meeting to be held at 8.15 p.m. on Tuesday, 10 September in the Physiology lecture theatre, Medical School, Observatory, Cape, at which addresses will be given by two of the distinguished overseas visitors to the South African Medical Congress. These are Dr. Ralph Tovell, of Hartford Hospital and Yale University, Hartford, Connecticut, past president of the American Society of Anesthesiologists and editor of '*Anesthesiology*', who will speak on 'Anaesthesia in the New World', and Dr. R. Bryce Smith, of the Nuffield Department of Anaesthesia, University of Oxford, who will speak on 'Anaesthesia in the Old World'.

At the meeting Dr. Tovell will present the Horace Wells Medal for 1956. This medal is awarded annually by the South African Society of Anaesthetists to the medical student of the University of Cape Town who achieves most distinction in the study of anaesthesia. It was at Hartford that Horace Wells pioneered the use of nitrous oxide as an anaesthetic.

Besides members of the Association, all members of the medical profession are invited to attend.

* * *

C.S.I.R. Bursaries. The Council for Scientific and Industrial Research invites applications for bursaries for work to be done in its own laboratories during 1958. Forms and regulations are obtainable from the Registrars of the South African Universities, or from the Secretary/Treasurer, C.S.I.R., P.O. Box 395, Pretoria. Applications should, where possible, be submitted through the registrars of the Universities to reach the Council not later than 31 August 1957. The following fields are included in those in which candidates will be required to work: The ecology of bilharzia-carrying snails in South Africa; Application of radio-isotopes; X-ray fluorescence with reference to techniques and applications; Technology and use of soya beans for human consumption; Protein chemistry; Electro-encephalography; Efficiency and productivity of native operatives; Treatment of industrial effluents; Measurement of air flows and other aerodynamic problems; Standards for special scientific, technical and medical libraries in South Africa; Environmental influences on mental and physical growth; Seaweed polysaccharides; Enzyme chemistry; Trace elements in nutrition; Biochemistry and biophysics in relations to human nutrition and metabolism; The bionomics of *Entamoeba histolytica;* The role of enzymes and hormones in human metabolism.

* * *

CIBA Foundation Awards, 1958. In order to provide further encouragement to research on problems of aging, the CIBA Foundation in London, an independent institution for the promotion of international cooperation in medical and chemical research, has organized another competition for the year 1958. The theme upon which candidates are invited to submit papers is entitled 'Basic Research Relevant to the Problems of Aging'. Approximately 5 awards, of an average of £300 each, are being offered. Entries are to be judged by an international panel of scientists, who will advise the Executive Council of the Foundation on their findings and will also have power to recommend variation in the size and number of the awards having regard to the standard of the entries received. The decisions of the Executive Council will be final. Entries must be submitted not later than 1 January 1958 to the Director, CIBA Foundation, 41 Portland Place, London, W.1. Where a contribution is the work of

more than one author, the name of the leading author should be stated first; the Executive Council will pay the amount of the award to the first-named author, leaving it to the authors to apportion the amount between themselves. The work submitted may be unpublished, may have been published in 1957, or may be under consideration for publication. If reprints in English are available, 10 copies should be provided. Papers may be in the candidate's own language and should not be more than 7,000 words in length, and in all cases a summary in English amounting in words to approximately 3% of the length of the paper must be attached. If the paper cannot be submitted in the form of a reprint, it must be typewritten, with double spacing and wide margins. Illustrations should then be on separate sheets, each with the author's name, and should be numbered; lettering and figuring upon them should be very clear. Legends should be typed on separate sheets. Papers submitted cannot be returned to their authors. The results of the competition will be announced in the scientific and lay press in July 1958.

REVIEWS OF BOOKS : BOEKRESENSIES

BLOOD-PRESSURE SOUNDS

Blood Pressure Sounds and their Meanings. By John Erskine Malcolm, B.Sc., M.B., Ch.B., F.R.C.S. Pp. vii + 93. 45 Figures. 12s. 6d. net. London: William Heinemann—Medical Books Ltd. 1957.

Contents: I. Introduction. II. The Elasticity of Arteries. III. The Electronic Co-ordinating Oscillometer. IV. The Oscillometric Curve. V. Volume Pressure Diagram of the Brachial Artery. VI. Relation of Korotkov's Sounds to the Oscillometric Displays. VII. Theory of Systemic Arterial Resonance. VIII. Stationary Wave Systems of the Brachial Artery. IX. Harmonic Analysis of the Brachial Arterial Pulse Wave. X. Turbulence, Cavitation and Water Hammer. XI. The Korotov Sounds. XII. The Visible Arterial Pulse. The Sigmoid Shape of Arteries. XIII. Determination of Nodes and Antidotes. XIV. Significance of the Terms 'Blood Pressure' and 'Flow Velocity'. XV. Implications. Growth and Metabolism. Arterial Damage. XVI. Implications (contd.). Persistent Stationary Waves. Arteriosclerosis. References. Index.

In this book the author analyses the physical basis underlying the production of Korotkov sounds heard at the brachial artery during the recording of the blood pressure. The literature is reviewed and the various theories discussed. An oscillometric recording system is employed. The harmonics of the stationary wave-systems of the brachial artery and systemic arterial resistance is discussed. The arterial system is broken into a series of nodes and antinodes. The fortuitous placing of the stethoscope at the brachial artery breaks up the upper-limb arterial system into a node at the brachial artery, so that waves set up in the obstructed artery become responsible for the mechanical production of the sounds.

The phenomena of turbulence and cavitation are described, with their influence on the production of the waterhammer phenomenon. The application of these principles to the Korotkov sounds is outlined. The visible pulsations in the arterial tree are explained on the basis of the theories of nodes and antinodes and the implication on the production of arterial damage, including arteriosclerosis, is outlined.

The book is so technical that it requires a special knowledge of physics and harmonics and is too complex for the average practitioner.

V.S.

EXPERIMENTAL PSYCHOPATHOLOGY

Experimental Psychopathology. Edited by Paul H. Hoch, M.D. and Joseph Zubin, Ph.D. The proceedings of the Forty-fifth Annual Meeting of the American Psychopathological Association, held in New York City, June 1955. Pp. x + 275. 86.50. New York and London: Grune & Stratton, Inc. 1957.

Contents: Foreword. Dedication. 1. Generalization and Extinction of Experimentally Induced Fear in Cats. *Norma A. Scheflen.* 2. Experimental Psychogenic Tachycardia. *W. Horsley Gantt and Ross A. Dykman.* 3. A Comparative Approach to the Experimental Analysis of Emotional Behavior. *Joseph V. Brady.* 4. Behavior and Metabolic Cycles in Animals and Man. *Curt P. Richter.* Discussion of Chapters 1-4. *William N. Schoenfeld.* 5. Experimentally Induced Depersonalization. *Otto von Mering, Kiyo Morimoto, Robert W. Hyde and Max Rinkel.* 6. An Investigation of the Psychopathologic Effect of Specific Emotions. *Oskar Diethelm and Frederick F. Flach.* 7. Disorganization: A Psychosomatic Principle. *D. Ewen Cameron.* 8. Perception of Parents and Social Attitudes. *D. H. Funkenstein, S. H. King and M. E. Drolette.* 9. Presidential Address: Concerning the Creative Process in Literature. *Merrill Moore.* 10. An Experiment in Inclusive Psychotherapy. *Hans Syz.* 11. Studies in Human Ecology: Factors Governing the Adaptation of Chinese Unable to Return to China. *Lawrence E. Hinkle, Jr., John W. Gittinger, Leo Goldberger, Adrian Ostfeld, Rhoda Metraux, Peter Richter, and Harold G. Wolff.* Discussion of Chapters 10-11. *Leo Alexander.* 12. Samuel W. Hamilton Award: Awareness, Attention and Physiology of the Brain Stem. *Stanley Cobb.* Introduction to Chapters 13-16. 13. The Problem of Schizophrenia in the Light of Experimental Psychiatry. *Paul H. Hoch.* 14. Use of Drugs in Psychodynamic Investigations. *James P. Cattell.* 15. Effects of Various Drugs on Clinical Psychopathology. *Harry H. Pennes.* 16. Experimental Investigation on Psychosurgical Patients. *Stanley Lesse.* Discussions of Chapters 13-16 *David McK. Rioch and Sidney Malitz.* Appendix: Members of the American Psychopathological Association. Index.

Something has happened in the world of experimental scientists which blunts their sensitivity toward both the Queen's and the President's English. Can it be that the tedium of chasing rats around cages or injecting drugs into medical students engenders this creaking verbiage? Or do they use this sterile prolixity as a dressing for the dry lettuce leaves of their thoughts?

It may be quite a common failing of reviewers to carefully pick a passage and then lay under it a charge of dynamite fused with the words 'chosen at random', but here is a sentence truly found at a casual opening of the book: 'These behavioural modifications are interpreted as a withdrawal of organismically rooted impulses from moralistic image formations with a coincident release of basic integrative assets.' This seems to mean simply: 'This behaviour seems to be due to a lessening of self-criticisms and a consequent release of basic forces.' Does one gain anything by calling a viewpoint a 'frame of reference' or a misfit a 'general adaptive misconstellation'?

The general vagueness behind these formidable brain-twisters comes out in other ways. A whole paper is seriously devoted to a test called the Brownfain test which clains to measure 25 'personality items' in a 'social attitude battery'. These 'values' include such entities as 'general culture', 'self acceptance' and 'overall adjustment'. Parched by this arid desert of words one turns hopefully to a chapter headed, 'Concerning the Creative Process in Literature'—alas, it must have been written for the rest of the contributors of this symposium. It contains such deep flashes of insight as 'There is no padding in the work of Homer; it is all hot stuff. Many scholars have noticed and mentioned this.'

And that fine classical note allows us to bring in Cicero, '*Quousque tandem abutere, Catalina, patientia nostra?*'

J. MacW. MacG.

ATLAS OF SKIN DISEASES

An Atlas of the Commoner Skin Diseases. 5th Edition. By Henry C. G. Wilson, M.A., D.M. (Oxon.), F.R.C.P. (Lond.). Revised with the collaboration of Harold T. H. Wilson, M.A., M.D. (Cantab.), M.R.C.P., D.T.M. Pp. viii + 375. 153 Colour Plates. 105s. post 1s. 6d. Bristol: John Wright & Sons Ltd., Medical Publishers. 1957.

Contents: I-V. Acne. VI. Alopecia Areata. VII. Cellulitis, Recurrent Streptococcal. VIII. Cheiropompholyx. IX. Chilblains. X. Dermatitis (Light Dermatitis). XI-XII. Dermatitis Artefacta. XIII.-XXI. Dermatitis, Contact Type. XXII. Dermatitis, Contact Type: Tar Melanosis, 'Mollusca', and Epithelioma. XXIII. Dermatitis, Generalized Exfoliative. XXIV. Dermatitis from Gold Injections. XXV. Dermatitis Herpetiformis. XXVI. Dermatitis, Infectious Eczematoid. XXVII-XXIX. Dermatitis Medicamentosa. XXX-XXXV. Dermatitis, Seborrhoeic. XXXVI. Eczema, Atopic. XXXVII-XXXVIII. Eczema, Infantile. XXXIX. Eczema (Nummular or Discoid) (Vesicular Stage). XL. Eczema, Vericose. XLI. Eczema (Patch Testing). XLII-XLIII. Epithelioma: Rodent Ulcer. XLIV. Erysipeloid. XLV. Erythema Induratum (Bazin's Disease). XLVI-XLVII. Erythema Multiforme (Iris). XLVIII. Erythema Nodosum. XLIX. Erythrocyanosis Crurum Puellarum L. Favux. LI. Glossitis, Chronic Superficial. LII. Granuloma Annulare. LIII. Granuloma Pyogenicum. LIV-LV. Herpes Zoster. LVI. Impetigo (Bockhart's). LVII. Impetigo (Bullous). LVIII. Impetigo Contagiosa. LIX. Intertrigo. LX. Keloidal and Hypertrophic Scarring. LXI-LXIV. Lichen Planus. LXV. Lichen Urticatus. LXVI-LXIX. Lupus Erythematosus. LXX-LXXI. Lupus Verrucosus. LXXII-LXXIV. Lupus Vulgaris. LXXV-LXXVI. Molluscum Contagiosum. LXXVII. Molluscum Sebaceum. LXXVIII. Moniliasis (Thrush). LXXIX-LXXXII. Naevus. LXXXIII-LXXXV. Neurodermatitis. LXXXVI. Paronychia. LXXXVII. Pityriasis Rosea. LXXXVIII. Pityriasis Versicolor. LXXXIX-XCVI. Psoriasis. XCVII. Rhinophyma. XCVIII. Rosacea. XCIX-CI. Scabies. CII. Sycosis Barbae. CIII-CXI. Syphilis. CXII. Tinea Barbae. CXIII-CXIV. Tinea Capitis. CXV. Tinea Circinata. CXVI. Tinea Cruris. CXVII-CXVIII. Tinea Pedis. CXIX. Pruritus Ani et Vulvae (et Scroti). CXX-CXXI. Tinea Unguium. CXXII. Urticaria. CXXIII. Varicose

Ulcer. CXXIV. Vitiligo (Acquired Leucoderma). CXXV-CXXVIII. Wart. CXXIX. Xanthoma Palpebrarum (Xanthelasma). CXXX. Xeroderma or Ichthyosis. CXXXI. X-ray Burn. CXXXII-CLIII. Some Less Common Skin Diseases. Index.

Appreciation of the shape, size and colour of a lesion is essential in diagnosing the more common dermatoses. Such visual appreciation can be learned from a good photograph. The advances of modern photography, well demonstrated in this atlas, permit reproductions of skin lesions so lifelike that they can be used as adequate substitutes for living clinical material for teaching purposes.

Semon and Moritz's Atlas was first produced in 1934; since then 4 further editions and 2 reprints have been issued, a testimonial to the demand and popularity of this work. In this 5th edition the text has been revised, the nomenclature modernized and 16 new plates added. Each plate is supported by short clinical lecturettes dealing with etiology, description, treatment and, where considered necessary, differential diagnosis. Dr. Semon, a doyen of British dermatology, is responsible for the text, which reveals both his clinical acumen and wide reading. His preference of mitigal to benzyl benzoate for scabies is certainly unusual. Surprisingly there is no mention of the use of local steroid applications in treating contact dermatitis.

The plates are superb and well selected. One would however have preferred Bazin's disease depicted on a calf and not on a shin; granuloma annulare and Rosenbach's erysipeloid would be better if pictured on the hands or fingers.

This atlas should be a useful addition to the general practitioner's library. The dermatologist will find it a valuable aid in clinical teaching.

J.J.J.

RADICULAR SYNDROMES

Radicular Syndromes—With Emphasis on Chest Pain Simulating Coronary Disease. By David Davis, B.S., M.D. Pp. 266. 69 Figures. $6.50. Chicago: The Year Book Publishers, Inc. 1957.

Contents: Section I. General Aspects of Cervico-Thoracic Root Syndromes. 1. Development of Concept of Root Syndromes. 2. Body Mechanics, Spinal Column, and Radicular Nerves. 3. Radicular Pain and the Segmental Pattern. 4. General Aspects of Symptomatology. 5. Diagnostic Signs. 6. Roentgenologic Aspects. *Section II. Lower Cervical and Upper Thoracic Root Syndromes with Chest Symptoms.* 7. Chest Pain of Nerve Root Origin. 8. Respiratory Manifestations of Root Compression Simulating Cardiac Asthma. 9. Clinical Significance of Tender Areas on Chest Wall. 10. Differential Diagnosis and Pitfalls. *Section III. Cervical Root Syndromes.* 11. Vertigo. 12. Occipital Headache. 13. Neck and Shoulder Girdle Syndromes. *Section IV. Treatment.* 14. Aspects of Medical Treatment. Exercises. Bibliography. Index.

The bulk of the material in this book deals with chest pain resulting from cervical and upper thoracic root syndromes which might be confused with pain of coronary origin. The author states in his preface that this book 'grew out of my interest in coronary disease and my mistakes in diagnosis during a period when I was largely unfamiliar with the role of root compression as a cause of chest pain'. In accepting a challenge there is ample proof in his book that he has made a very systematic and comprehensive study of the problem of radicular pain.

In the earlier sections he deals with the normal anatomy and the body mechanics of the spinal column, and the relationship of the nerve roots to the skeletal structures in the normal as well as in pathologic conditions involving the spinal column at its various levels.

A most commendable feature of this book is the clear differentiation between pain of dorsal root and pain of ventral root origin. Herein lies a better understanding of and a more rational therapeutic approach to the various types of chest wall pain which may mimic anginal pain. He cites Frykholm's observations in humans, indicating that pain arising from the ventral root may be severe or dull, but is essentially boring or aching in character; it differs from the sharp radiating pain of dorsal nerve root irritation, which furthermore is often associated with parasthesias in the distribution of the involved posterior root. It is his contention that painful and tender areas in the chest wall which often are attributed to local causes such as fibrositis or costo-chondral arthralgia, are indeed indicative of ventral root irritation, the commonest cause of which is osteo-arthritis and disc degeneration.

He places much reliance on reproducing painful syndromes during the course of the examination of the patient, and proceeds to describe clearly the manoeuvres he applies to do so. He further places much reliance on tender areas in the chest as evidence of root irritation, and emphasizes that an absence of such tender areas is against their being caused by root irritation; and finally he relies on the response to traction, both as a diagnostic aid and for therapeutic purposes.

Throughout the various sections of the book he cites case reports which illustrate these various points, and repeatedly emphasizes the fact that many patients have both radicular pain and a pain of coronary insufficiency.

There is an interesting section dealing with vertigo in association with the sub-occipital headache, which in his opinion is caused by upper cervical root compression (C2 C3). The anatomic basis for this claim lies in the fact that the medial longitudinal bundle which carries proprioceptive impulses, extends down into the upper cervical cord. Nerve root pressure setting up a bombardment of afferent impulses to the cervical cord could understandably disturb this primary coordinating system and cause vertigo. Nystagmus is characteristically absent in this syndrome, proving that it is not vestibular in origin.

The final part of this book deals with the physiotherapeutic and rehabilitating aspects of pain resulting from root compression.

This book can be read with profit by a wide variety of physicians, regardless of their primary field of interest.

T.J.D.

PSYCHOTHERAPY BY THE GENERAL PRACTITIONER

The Doctor, His Patient and the Illness. By Michael Balint, M.D. Pp. x + 355. 40s. net. London: Pitman Medical Publishing Co. Ltd. 1957.

Contents: Preface. I. Introductory. *Part I: Diagnosis.* II. The General Problem. III. The Patient's Offers and the Doctor's Responses. IV. Elimination by Appropriate Phsycial Examinations. V. Incidence and Evaluation of Neurotic Symptoms. VI. Level of Diagnosis. VII. The Collusion of Anonymity. VIII. The General Practitioner and his Consultants. IX. The Perpetuation of the Teacher-Pupil Relationship. *Part II: Psychotherapy.* X. Advice and Reassurance. XI. 'How to Start'. XII. 'When to Stop'. XIII. The Special Psychological Atmosphere of General Practice. XIV. The General Practitioner as Psychotherapist—A: Two Illustrative Cases. XV. The General Practitioner as Psychotherapist—B: A Difficult Case. *Part III: General Conclusions.* XVI. The Apostolic Function—I. XVII. The Apostolic Function—II. XVIII. The Doctor and His Patient. XIX. The Patient and his Illness. XX. General Practitioner Psychotherapy. XXI. Summary and Future Outlook. Appendix I. Training. Appendix II. Selection. Appendix III. Follow-up Reports. Appendix IV. An Additional Role for the Psychological Clinic by J. D. Sutherland. References to Case Material. References to Doctors Participating. Index.

This is a book about psychomatic medicine It is calculated that at least 40% of patients who find their way into the general practitioner's waiting room are psycho-somatic cases. The author is of opinion that the responsibility for treating such cases from the psychical as well as the somatic point of view lies with the general practitioner, because it is he who knows the patient's background, his family life, his financial problems and his personality in a way that no specialist can.

The problem is how the general practitioner is to be trained to meet this responsibility. Certainly his medical course gives him no training for it at all. At the Tavistock Clinic, Dr. Balint, a psychiatrist, made an experiment in group training: doctors voluntarily joined the group and submitted the histories of their cases and their own attempts to their colleagues for criticism and suggestions. The author admits that he met with indifferent success, but one feels that here is the only method of training which can succeed.

One difficulty is that every doctor has his own particular mode of practice, which he endeavours to get his patients to accept—what the author calls the 'apostolic function' of the doctor. Another difficulty, of course, is lack of time; the investigation of the causation of psychical disturbance may be, and generally is, time-consuming. Numerous examples are given of case histories with varying modes of approach. A large percentage of the doctors involved did not stay the course and the successes obtained were not numerous. One can only echo the author's plea for more research into this vitally important question.

I think there is much point in the author's views on the general practitioner's relation to his consultants and his deprecation of the continuation of the teacher-pupil relationship; the general practitioner must regard patients as *his* responsibility, whilst calling in consultants' help where necessity arises.

The 'collusion of anonymity' to which the author refers is a very real danger, especially in our teaching hospitals, where the patient seldom knows which of several doctors in a firm is *his* doctor.

Finally he stresses the importance of 'listening' to the patient as opposed to asking a lot of quesions.

Altogether this is a book well worth reading.

F.R.L.

RECTAL SURGERY

Surgery of the Anus, Anal Canal and Rectum. By E. S. R. Hughes. Pp. xi + 304. 276 Figures. 50s. net + 1s. 4d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1957.

Contents: I. Surgical Anatomy of the Anal Canal and Rectum. II. The Symptoms of Ano-Rectal Disease. III. The Investigation of Ano-Rectal Disease. IV. Surgical Wounds of the Anal Region. V. Anaesthesia for Ano-Rectal Surgery. VI. Ano-Rectal Suppuration. VII. Anal Fistula (Fistula-in-Ano). VIII. Sinuses Related to the Anus. IX. Anal Fissure (Fissure-in-Ano). X. Haemorrhoids. XI. Prolapse of the Rectum. XII. Pruritus Ani. XIII. Proctitis (Non-Specific). XIV. Benign Tumours of the Rectum. XV. Carcinoma of the Rectum. XVI. Squamous Cell Carcinoma of the Anus and Anal Canal. Historical Appendix. Index.

This book is the outcome of vast practical experience in the art of proctology. The facts are presented with simplicity, clarity and clear illustrations. In places these illustrations are diagrammatic, which sometimes tends to oversimplification. The book is up to date as is shown by the description of the anatomy of the anal sphincters. It is interesting to note in this respect how 'Hilton's line' is rapidly becoming a relic of the past in modern proctological descriptions.

The chapters dealing with the subject of skin cover in relation to anal wounds are the feature of this book. The skin defects produced by fistula and fissure operations are treated by either primary or delayed skin grafts—a method for which the author has become well known. The problem of skin-grafting anal wounds was something which taxed the mind of Mr. W. Gabriel as long ago as 1927, when this subject was presented to the Royal Society of Medicine, and strangely enough it has never really been condemned. Even the now popular internal anal sphincterotomy operation for fissure is not preferred to the more orthodox method *plus* skin graft. These controversial points are always welcome, particularly when presented with such enthusiasm.

A sound method of treatment seems to be the operation described for rectal prolapse (synchronous combined abdomino-perineal repair). However, no figures are presented to back up the efficiency of this manoeuvre. Almost a third of the book is reserved for a detailed description of cancer of the rectum.

This book should prove a valuable asset to surgical postgraduate students and surgeons.

P.H.

UROLOGY

General Urology. By Donald R. Smith, M.D. Pp. 328. Illustrations. Los Altos: Lange Medical Publications. 1957.

Contents: 1. Anatomy of the Genitourinary Tract. 2. Symptoms of Disorders of the Genitourinary Tract. 3. Physical Examination of the Genitourinary Tract. 4. Urologic Laboratory Examination. 5. Roentgenographic Examination of the Urinary Tract. 6. Instrumental Examination of the Urinary Tract. 7. Urinary Obstruction and Stasis. 8. Nonspecific Infections of the Urinary Tract. 9. Specific Infections of the Urinary Tract. 10. Urologic Aspects of Venereal Diseases in the Male. 11. Urinary Stones . 12. Injuries to the Genitourinary Tract. 13. Tumours of the Genitourinary Tract. 14. The Neurogenic Bladder. 15. Disorders of the Perirenal Area. 16. Disorders of the Kidneys. 17. Disorders of the Ureters. 18. Disorders of the Bladder, Prostate, and Seminal Vesicles. 19. Disorders of the Penis and Male Urethra. 20. Disorders of the Female Urethra. 21. Disorders of the Testis, Scrotum, and Spermatic Cord. 22. Intersexuality. 23. Renal Hypertension. 24. Infertility. 25. Psychosomatic Urologic Syndromes. Appendix: Index.

As Professor Donald R. Smith states, this book has been written for the student and the general practitioner. It serves that purpose admirably. It is well written and gives a concise and at the same time fairly comprehensive and up-to-date account of the speciality. The approach is mainly a clinical one and the chapters on general examination and investigation are very good. There is also an interesting chapter on psychosomatic involvement of the urinary tract.

The diagrams are good, but this does not apply to the X-ray illustrations, which are mainly too poor to serve any useful purpose. In the section on anatomy the scrotum is erroneously described as having fat beneath its dartos muscle layer. The illustration of the autonomic nervous system shows the testis to have an afferent nerve supply passing to S2, 3 and 4 instead of D10, 11 and 12 and L1 segments of the cord. It is rather astonishing to read of aborting an attack of acute epididymitis by injecting 1% Novocain into the spermatic cord. The description of schistosomiasis holds good for the heavy infestation found in Egypt, but does not apply to the much milder type of infection commonly found in Africa south of the equator.

In describing the complications of injury to the kidney, secondary haemorrhage has been omitted. Perforation of the ureter by a catheter during retrograde procedures is not a very uncommon complication and does not call for open drainage. Observation is usually all that is necessary.

The book has a good index and bibliography and can be strongly recommended both to the student and to the general practitioner.

S.S.

CORRESPONDENCE : BRIEWERUBRIEK

THE DURBAN CONGRESS

To the Editor: For me one of the outstanding attractions of the forthcoming Durban Congress is the prospect of hearing Sir Russell Brain speak, for few can rival his knowledge, wisdom and clarity of thought and expression. I wish, however, that the organizers of the Congress would not persist in describing him as President of the Royal College of Physicians of London, which august position has for some months been held by Prof. Robert Platt, who made a memorable visit to South Africa not very long ago. Prof. Robert Platt has been for years the Professor of Medicine at Manchester.

It may have escaped the notice of your readers that the immediate Past-president of the Royal College of Surgeons of England, Prof. Sir Harry Platt, who is coming to Durban, has been for years the Professor of Orthopaedic Surgery at Manchester.

All of which brings me to my point, namely, that the Royal Colleges by their choice of Presidents have strikingly vindicated the conviction, firmly held by all good Mancunians, that what Manchester thinks today the rest of the world thinks tomorrow!

G. R. Crawshaw

Bulawayo
6 August 1957

THE WATER-HAMMER PULSE

To the Editor: I am amazed that almost without exception our teachers—and subsequently their students—when asked what a 'water-hammer' is, when referring to the Corrigan type of pulse, insist on a vague reference to some form of mechanical pounding machine operated by water, or even—as I was once informed by a very senior lecturer—a form of toy operated by connecting it to a tap'.

Having come to regard with some awe the medical man's accuracy in likening things medical to everyday objects (especially foodstuffs) I was, as a student always perplexed that a pulse—which is after all an impulse transmitted by fluid in a hollow vessel—could be likened to anything resembling some wondrous form of trip-hammer.

Reference to any good dictionary however, will reveal that a 'water-hammer' has nothing whatsoever to do with hammers as we know them in any form, but is defined as 'the percussion wave felt in a pipe containing water when a tap is suddenly opened or closed'.

We have all at one time or another heard such a percussion wave, especially when a pipe is loose. If one happens to have one's hand on the pipe during the percussion the description 'water-hammer pulse' at once becomes apparent, as also the similarity of effect due to fluids in hollow vessels.

I hope that this will clear a lot of confusion in the minds of students and others and restore their faith in the medical man's genius for analogy and simile.

D. J. W. Kinnear

West Rand
Transvaal
17 August 1957